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ABSTRACT

Explored are children's vulnerability and the pediatrician's role in relation to the problems posed by air pollution. Research is noted to have included a search of biomedical literature over the past 10 years; attendance at medical meetings; conferences with air pollution researchers, environmental protection administrators, and specialists in various branches of pediatrics; and scheduled interviews with a representative sample of active practitioners. Chapters address the following topics (sample subtopics are in parentheses): introduction (children as convenient epidemiologic subjects); outdoor pollutants (major problems of measurement and attempts at quantifying meaningful estimates); contaminated indoor air (principal indoor offenders and preventive measures); the effects of airborne irritants/toxins on children (mental illness and behavior changes); the concern of practicing pediatricians (air pollution in the pediatric literature and interviews with public health officers); and the environment and preventive pediatrics (benefits of updating the prevention concept and environmental and child-oriented groups). Appended are tables on pollution studies, interview materials, a paper on air pollution and childhood lower respiratory disease, and a paper on pollution and the ambient air of small children. (SBH)

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CHILDREN, PEDIATRICIANS, AND POLLUTED AIR

By

Dorothy Noyes Kane

B.A. Goddard College, 1964

M.P.H., M.U.S. Yale University, 1966

Robert W. Kates

Robert W. Kates, Ph.D., Advisor
University Professor
Clark University

A Dissertation Submitted in Partial Fulfillment of
The Requirements for the Degree of
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ABSTRACT

Children and pediatricians are the foci of attention in this exploration into the problems posed by air pollution: children because of their unique vulnerabilities; pediatricians because of their unique responsibilites. Who is at special risk to what pollutants--when, where, how? How is it evaluated? Who is concerned about this risk--why, or why not? These are some of the questions which this exercise attempts to probe.

Several methodologies have been employed: a comprehensive search of the world's biomedical literature for the past 10 years (longer for the pediatric literature); attendance at medical meetings; conferences with air pollution researchers, environmental protection administrators, specialists in various branches of pediatrics; and scheduled interviews with a representative sample of active practitioners.

In addition to a review of research dealing with the many ways in which children (including embryos and fetuses) are or may be affected by exposure to chronic low levels of polluted community air, reports of the dangers of contaminated indoor air are discussed.

The matter of concern for the problem is dealt

with at some length--a concern felt by some practitioners (the "Mind-the-Iceberg" type), but apparently not shared, certainly not well articulated, by official pediatric groups ("Count-the-Bodies" people).

This study concludes on a positive note. Since pediatricians, of all physicians, have long been distinguished by their prevention orientation, and since polluted air poses so many potential long-term threats, it is suggested that an update of the concept of prevention is very much in order. This to help insure optimal health and well-being and greater productivity over a lifetime for a very sizeable minority of the U.S. population. How such an update can be accomplished by both individuals and groups in the clinical, teaching, and research communities is the subject of the concluding chapter.

TABLE OF CONTENTS

PREFACE. x

Chapter

I. INTRODUCTION. 1

Children as Convenient Epidemiologic Subjects. . 1
Why Children Require Special Consideration . . . 3
Higher inspiratory flow rate at best 4
Higher level of activity 4
Closer proximity of breathing zone to the ground 5
Nature of play activity. 7
More mouth breathing 7
More frequent respiratory tract infections . . 8
Age-specific developmental factors 8

II. WHO'S AT RISK TO HOW MUCH OF WHICH OUTDOOR POLLUTANTS? 11

Major Problems of Measurement. 12
Ambient air standards. 12
Aerometric data. 14
Variations in susceptibility 16
Attempts at Quantifying Meaningful Estimates . . 17
APHA estimates 18
EPA estimates. 20
A stratified risk estimate 25

III. CONTAMINATED INDOOR AIR ALSO A RISK 31

Principal Indoor Offenders 32
Tobacco smoke. 32
Aerosol sprays 38
Heating, ventilating, and cooking equipment. . 41
Housedust. 43
Preventive Measures. 45

IV. HOW CHILDREN ARE (AND MAY BE) AFFECTED BY AIRBORNE IRRITANTS/TOXINS 48

Pollution Vulnerability Index. 49
Non-Allergic Airways Disease 50
Pulmonary function 52
Acute respiratory illness. 60
Effects of sulfur oxides 61
Effects of nitrogen oxides 70
Effects of oxidants. 73
Chronic respiratory disease. 76
Allergic Airways Disease: Asthma 78
Mental Illness and Behavior Changes. 90
Low level lead poisoning 94
Wide prevalence. 96
Reduction of "undue" absorption level. . . . 99
Airborne lead in dust and dirt 101
Neuropsychologic manifestations of low lead levels. 105
Commentary 109
Growth and Development 111
Cancer, Mutations, and Malformations 118

V. ARE PRACTICING PEDIATRICIANS CONCERNED ABOUT POLLUTED AIR? 123

Air Pollution in the Pediatric Literature. . . . 124
What might have been learned from the literature 126
What Active Practitioners Think, Feel, and Do. . 129
Interview procedure. 130
Sociologic profile 132
Knowledge and its source 133
Attitude toward recent evidence. 135
Action taken with respect to pollution 137
How Concerned Are Connecticut Pediatricians? . . 139
Measures of concern. 140
"Mind-the-Iceberg" type 142
"Count-the-Bodies" type 144
Interviews with Public Health Officers 146
Commentary 148

VI. THE ENVIRONMENT AND PREVENTIVE PEDIATRICS 156

Benefits of Updating the Prevention Concept. . . 156
Principles and Parameters of an Updated Prevention Program 160
What Individual Practitioners Can Do 164
What Organized Pediatrics Can Do 168
What the Research Community Can Do 170
What Medical Schools Can Do. 172
What Environmental and Child-Oriented Groups Can Do 173

APPENDICES. 175

Appendix A: Ventilatory Function Studies. . . . 175

Appendix B: Low Level Lead Poisoning. 177
B-1: Prevalence Studies. 177
B-2: Animal Studies. 183
B-3: Human Studies 185

Appendix C: Growth and Development Studies. . . 190

Appendix D: Interview Materials 194
D-1: Questionnaire 194
D-1.1: Exhibit 197
D-1.2: Exhibit 217
D-1.3: Exhibit 238
D-2: Letter and Areas Covered. 243
D-3: AMA Guide 246

Appendix E: AAAS Presentation 247

ABBREVIATIONS 259

LITERATURE CITED. 260

ILLUSTRATIONS

Figure

1. Polluted Outdoor Air. 6

2. Contaminated Indoor Air 33

3. Estimate of Lower Respiratory Disease and Suspended SO_4 Concentration 68

4. "Mind-the-Iceberg" Type 143

5. "Count-the-Bodies" Type 145

LIST OF TABLES

1. National Ambient Air Quality Standards 13
2. EPA's At-Risk Estimates. 25
3. Stratified Risk Estimate 29
4. Pollution Vulnerability Index. 50
5. Effects of NO_2 on Acute Respiratory Disease. 71
6. Views re: Impact of Air Pollution. 132
7. Source of Information re: Air Pollution. 134
8. Measures of Concern for Air Pollution. 141
9. Pediatrician Ratings of Concern 141

PREFACE

It was Barry Commoner at an American Public Health Association (APHA) meeting in the late 1960's--in pointing to the present generation of youngsters as the first to bear the brunt of strontium-90 in their bones (as a result of radioactive fallout)--who first aroused my concern for the implications of 20th century environmental hazards in connection with children's health and wellbeing. This concern deepened as a health educator at Southern Connecticut State College, and triggered their first Environmental Sciences Institute in 1970, and their Environmental Studies Center a year later.

Upon terminating my association with the field of graduate education and resuming full-time work as a writer in 1973, this concern took the form of further research in preparation for a book for lay consumption.

Recognizing the imperative for assistance in sensitively assessing the plethora of data pouring forth from diverse sources and disciplines in the burgeoning field of environmental health--as well as the value (and hopefully the credibility) to be gained from acquiring additional research expertise at a higher level--the

decision was made to postpone further work on the book in order to pursue additional investigations along several lines.

What eventuated after a reasonably prolonged gestational period of thought and consultation is a three-pronged investigative project aimed at learning more not only about the impact of air pollution on children's health and wellbeing, but upon pediatricians' knowledge and perception of this, including their views of where their responsibilities lie with respect to upgrading air quality. This research journey might be likened in some respects to the opening of Pandora's box, for it led down many pathways besides those usually thought of as health-related: to socioeconomic and political considerations, and psychological, philosophical, and ideological considerations as well--as a perusal of this presentation will indicate.

The three-pronged approach consisted of the following: (1) search for certain domestic and foreign literature over an extended period relating to the subject of air pollution; (2) conferences with air pollution researchers on the west and east coasts; and (3) interviews with practicing pediatricians and public health officers in the State of Connecticut.

Search of the Literature

This search pursued at the Yale University medical library covered the following: (1) American pediatric literature from 1950-1974 (as delineated in Chapter V); (2) American professional periodicals and books, including educational, environmental, and biomedical (other than pediatric) publications from the late 1960's-1974, and foreign literature from environmental and biomedical (including pediatric) publications for the same period for which English translations or abstracts were available; (3) U.S. Government documents, including National Academy of Science-National Research Council (NAS-NRC) reports prepared for the Senate's Committee on Public Works, U.S. Environmental Protection Agency (EPA) community air pollution studies, and annual reports of the President's Council on Environmental Quality (CEQ).

Contacts with Researchers

In addition to the search of the literature, background material for the dissertation included a number of country-wide trips, as follows: (1) to San Francisco to attend the American Medical Association's (AMA) Air Pollution Medical Research Conference held 5-6 December 1974; to confer there with the Chairman of the American Academy

of Pediatrics' (AAP) Committee on Environmental Hazards, with the Head of the Environmental Epidemiology Unit of the California Department of Public Health, and with EPA and other epidemiologists and researchers attending the conference; (2) to Research Triangle Park, Durham, N.C. in June 1975 to meet with the Director and various staff members of the Human Studies Laboratory at the National Environmental Research Center (NERC); (3) to New York City in October 1974 to confer with a member of AAP's Committee on Environmental Hazards; (4) to Boston also in October 1974 to confer with the Chairman of AAP's Massachusetts Chapter of the Committee on Environmental Hazards; and (5) to Boston again in February 1976 to participate in a colloquium (as explained later). The work included also a number of telephone "interviews" and consultations with out-of-town researchers.

Interviews With Practicing Pediatricians

A third facet of fact-gathering in preparation for this presentation is field investigations involving personal interviews with more than 40 practitioners of clinical pediatrics and six public health officers in 13 cities and towns in the State of Connecticut (as described in Chapter V). This involved a number of consultations with the Department of Environmental Protection (DEP) in

Connecticut to determine which communities should be considered "Dirty Air" and which "Clean Air" areas. To interview pediatricians at their convenience necessitated 43 out-of-town trips; the total elapsed time for completing the interviews was three months.

Acknowledgements

To all the busy people (including many pediatric practitioners and public health officers in the State of Connecticut) who gave quite unstintingly of their time and expertise in exploring this problem with me, I am deeply grateful. I feel especially indebted to several who went far beyond any call of duty to examine in considerable detail, and criticize certain sections of this presentation: namely John H. Knelson of NERC's Health Effects Laboratory (Airways Disease); Herbert L. Needleman of Harvard Medical School (Mental Ills); Michael I. Cohen of Albert Einstein College of Medicine (Growth and Development); and Nathan Lerner of Southern Connecticut State College (chemistry matters).

Over a 20-month period, under the unbelievably patient guidance and inspiring tutelage of Robert W. Kates of Clark University, I have arrived at a greater understanding not only of myself and of what makes me tick, but of how a sophisticatedly concerned person would

pursue this matter of concern, and of the extensive parameters and incredible minutiae involved in achieving a valid assessment of the pediatric implications of airborne pollutants. The lessons I have learned at his expense and the sacrifice of his time and energy (and of his wife's, too) are too numerous to delineate, but they are invaluable. (Perhaps the most important of these lessons is the futility of the search for absolute truth or of a final indisputable authority.)

Appreciated also in the opportunity to join in a colloquium under his arrangement at the 142nd annual meeting of the American Association for the Advancement of Science in Boston on 22 February 1976, with a presentation on "Pollution and the Ambient Air of Small Children" (see Appendix E).

Only the tip of the iceberg of understanding, I surmise, is visible to me now; my hope is that the magnitude of the insights which are bound to emerge little by little as the result of Dr. Kates' so-called "gentle but tough" treatment will prove not overwhelming but highly stimulating as time goes on.

CHAPTER I

INTRODUCTION

Children as Convenient Epidemiologic Subjects

Traditionally, epidemiologists engaged in air pollution research have thought of children as representing more ideal subjects than most adults because of their freedom from a number of the variables which complicate the problem of identifying the factor(s) responsible for causing or aggravating health (or disease) effects in humans. Thus, a number of official health/environmental groups have described the specific advantages of air pollution studies of children. For example:

(1) American Academy of Pediatrics (AAP 1970):

> Children as a group may serve to delineate more clearly the effect of air pollution on health. They (1) spend most of their lives and attend school in limited geographical areas where air quality and meteorological effects can be measured and school absenteeism may also be ascertained, (2) are more susceptible to most respiratory infections, (3) are not largely affected by smoking because it becomes a factor in only a small sub-group, and (4) are not exposed to the occupational hazards to which many adults are exposed.

(2) U.S. Environmental Protection Agency (EPA, Nelson et al. 1974):

> Children provide a unique opportunity to assess the effect of air pollutants on the respiratory tract. The young are vulnerable to acute respiratory

disease. Unlike adults, children have not usually been subjected to self-pollution by cigarette smoking, or to a bewildering series of residential changes. Any of these factors can profoundly confound needed estimates of past exposure to ambient air pollutants.

(3) National Institute of Environmental Health Sciences (NIEHS, Rall 1974):

> A number of studies have been carried out on children, who are particularly suitable because they tend not to smoke cigarettes and for the most part do not engage in dusty jobs. They also may be especially sensitive to the effects of pollution.

(4) National Academy of Sciences (B. Goldstein 1975a):

> Children represent a useful group to study, in that their lesser mobility produces a more homogeneous pollution exposure and the effects of cigarette smoking and occupational exposure are minimized.

Only recently has it become obvious that children are highly important candidates for air pollution research--not simply because they represent easier, more convenient epidemiologic subjects--but for two very basic reasons: they would appear to be especially sensitive to insults from air pollution, and the sequelae of such insults may have a greater and longer-term pathologic impact.

In a recent Task Force report, the National Heart and Lung Institute (NHLI 1972) observed that:

> There are scattered data which suggests that the initiation of environmental lung disease as a result of air pollution in childhood may be the starting point for continuation and progression of such disease in later life. . . . Should such a relationship be established between exposure to polluted air in childhood and subsequent increased risk of chronic lung

disease, it will have identified an important point for the application of preventive measures.

Of course, it isn't only children's lungs that may be adversely affected by air pollutants. A number of other organs and systems--such as the central nervous system (CNS)--may also bear the brunt of toxic agents (though evidence of the ill effects may not become manifest for years or decades), as will be described in subsequent sections of this study.

It is the chronic effects from low level pollution--not the acute morbidity and mortality arising from the more dramatic and severe air pollution episodes--which is the concern of this presentation.

Why Children Require Special Consideration

What makes children more susceptible to irritation and/or toxicity from air pollutants than most adults? There are certain distinctions which set them apart from adults--anatomic, physiologic, biochemical, and behavioral. These distinctions play a role with respect to the difficulties experienced by many children in coping with, and adapting to, the noxious particles, gases, and vapors in the air they breathe:

(1) higher inspiratory flow rate at rest,

(2) higher level of activity,

(3) closer proximity of breathing zone to the

ground,

(4) nature of play activity,

(5) more mouth breathing,

(6) more frequent respiratory tract infections, and

(7) age-specific developmental factors.

Each of these will be discussed briefly in turn.

Higher Inspiratory Flow Rate at Rest

Children are known to have a higher resting metabolic rate and ventilation (Knelson 1974a) and relatively greater tidal volume than adults (NAS 1975). Thus, per unit of body weight, the young breathe in more air (and thus more air pollutants) than older persons. Indeed, under comparable exposure, it is said that children inhale two to three times as much of a pollutant per unit body weight as do adults (Lin-Fu 1975). This is of significance, since the efficiency of removal of pollutants would be expected to depend to some extent on the rate of flow into the lung (NAS 1975).

Higher Level of Activity

Added to the metabolic difference between infant or child and adult, is the propensity of the young for physical activity. As the level of activity rises, so too does the inspiratory flow rate--and in a roughly exponential

manner. In fact, during exercise, pulmonary ventilation can increase by a factor of about 10 (NAS 1975). The very biologic and behavioral nature of childhood thus places this age group in a position of heightened susceptibility to inimical aerosols--not only in the upper airways, but penetrating more deeply into the lung.

Closer Proximity of Breathing Zone to Ground

Since gravity forces many heavier air pollutants to the ground (or floor)--and some of these (like the metals) may possess the most damaging properties as far as developing organisms are concerned--small children by virtue of their stature are nearer to, and thus more subject to heavier attack from, a potential source of what for them can prove to be serious pathology. Dustfall laden with neurotoxic and otherwise deleterious particles can add substantially to a child's total respirable intake (Figure 1).

In the case of lead, the concentration in ambient air varies inversely with altitude, and devices used in measuring air lead concentration are rarely, if ever, placed at the height at which young children breathe. Lead concentration at 1.5 meters in one study was twice that at 20 meters above the ground (Lin-Fu 1975). Similarly, the concentration of ozone is greater at a lower

Fig. 1. Polluted outdoor air from many sources

level. In fact, one study showed the concentration of ozone to be 4 times greater at 1 than at 2 meters (Turner et al. 1974).

Nature of Play Activity

Coupled with the promixity of their breathing zone to a greater concentration of the heavier and in some cases potentially more devastating particulates is the dust-raising proclivity of small (and often large) children's play activity in house, yard, or school. Feet in almost constant motion, supplemented by sticks and balls and similar objects, serve to augment the invasion potential of near-the-ground pollutants into the respiratory tract (and often the alimentary tract as well). Hands contaminated with dust and dirt also contribute to a small child's total intake of airborne particles, particularly those addicted to the pica habit (a habit of ingesting non-nutritious items of various sorts). This makes for a potentially dangerous situation with respect to lead (and possibly other metals as well), since in children (in contrast to adults) there is apparently greater absorption through the gastrointestinal tract (Lin-Fu 1973).

More Mouth Breathing

More exercise plus blockage of the nasal passageways

can both lead to an increase in mouth breathing, which in turn leads to less effective filtering of respirable air pollutants. For these two reasons--since they are more physically active and since the integrity of their respiratory defense mechanism is more often impaired than adults'--children are frequently less able to adjust to or handle successfully the effects of inhaled toxic aerosols.

More Frequent Respiratory Tract Infections

A major contributor to blockage of the nasal passageways--and thus to impairment of the normal filtering process--is upper respiratory infection, which accounts for a large proportion of the acute illnesses of childhood. With the lung sensitized by viral infection, noxious air pollutants--because of their suppressive effect on phagocytosis and on antibody formation (and thus on the body's defense mechanism)--may prove a dual threat: (1) compounding the risk of secondary bacterial infection in the lung, and (2) impairing the development of effective immunity to viral infection as well (Green 1974).

Age-Specific Developmental Factors

From the first trimester in utero and throughout the entire period of gestation, at birth and during the newborn period, infancy, childhood, and into puberty and

adolescence, developing organisms are at special risk from certain airborne agents. Many different organs, systems and subsystems (primarily perhaps the immune, hematopoietic, skeletal, and CNS) have proved, and many more may prove to be, highly vulnerable target areas--at various times and in various ways, depending both upon the nature and extent of the exposure as well as upon the infant or child's maturational or nutritional state.

As one example, inadequacies of certain minerals, vitamins, and proteins may potentiate the toxic effects of lead. Further, the predilection of the young for harboring this metal in the soft--as contrasted with the dense bone--tissue may be another factor which makes children more vulnerable to deleterious sequelae from this pollutant.

Many chemicals are known to pass the placental barrier--thus imposing mutagenic, teratogenic, carcinogenic, and other threats to the fetus. Maternal exposure, therefore, is highly relevant in connection, first, with modifying the functions of specific organs and systems in the infant; and, second, in the ability of the infant to cope at the time of birth (and later) with any toxic agent(s) in his tissues.

Thus, for these seven reasons, children should no

longer be considered merely convenient epidemiologic subjects. As distinctly unique psychobiologic entities, they deserve special consideration because of their vulnerability to the deleterious impact from air pollutants. At their play--which is their work--they, too (and not just adults) risk occupational hazards both indoor and out, as will be described further in subsequent chapters.

CHAPTER II

WHO'S AT RISK TO HOW MUCH OF WHICH OUTDOOR POLLUTANTS?

Though children are by nature and behavior and environment peculiarly vulnerable to air pollution, we perceive only very crudely the population at risk. As few as a couple of million or as many as 60 million (all children under 14 years of age) may be subject to levels of pollution capable in time of producing at least troublesome, if not downright disabling or seriously crippling, chronic disturbances.

Such estimates as have been made have two basic limitations. They have dealt only with the risks associated with disorders of the respiratory tract; they have not included potential or actual risks to any other systems or organs of the body, as just mentioned. Therefore, they provide only a very limited picture of morbidity consequences of polluted air. The other limitation of these estimates concerns the number of pollutants considered; only a small fraction of the pollution scenario has been examined, as will be explained.

Major Problems of Measurement

To understand why the reliability and significance of such estimates as have been made are in question, let us consider three major problems in providing better estimates. This can perhaps best be understood if we look at the following: (1) ambient air standards; (2) aerometric data; and (3) variations in susceptibility.

Ambient Air Standards

Estimates have been based on one or more of six pollutants for which ambient air quality standards have been set (a bureaucratic necessity, perhaps, but in reality a biologic absurdity for reasons about to be supplied). These six are: sulfur dioxide (SO_2), particulates (TSP), photochemical oxidants, hydrocarbons, carbon monoxide (CO), and nitrogen dioxide (NO_2)--see Table 1.

These six pollutants represent only a small fraction of the thousands of pollutants in ambient air, a heavy proportion of which--perhaps even 50-75%--have not yet been identified. Even these six pollutants are not all simple single chemical elements; they are mostly mixtures--yet the index used as the standard (like SO_2 for all the sulfur oxides and ozone for all the photochemical oxidants) does not accurately reflect the properties of the agents for which they act as surrogates. They do not

TABLE 1

NATIONAL PRIMARY AND SECONDARY AMBIENT
AIR QUALITY STANDARDS

Pollutant	Type of Standard	Averaging Time	Frequency Parameter	Concentration	
				ug/m^3	ppm
Carbon monoxide	Primary and secondary	1 hr	Annual maximum[a]	40,000	35
		8 hr	Annual maximum	10,000	9
Hydrocarbons (nonmethane)	Primary and secondary	3 hr (6-9 a.m.)	Annual maximum	160[b]	0.24[b]
Nitrogen dioxide	Primary and secondary	1 yr	Arithmetic mean	100	0.05
Photochemical oxidants	Primary and secondary	1 hr	Annual maximum	160	0.08
Particulate matter	Primary	24 hr	Annual maximum	260	--
		24 hr	Annual geometric mean	75	--
	Secondary	24 hr	Annual maximum	150	--
		24 hr	Annual geometric mean	60[c]	--
Sulfur dioxide	Primary	24 hr	Annual maximum	365	0.14
		1 yr	Arithmetic mean	80	0.03
	Secondary	3 hr	Annual maximum	1,300	0.5

SOURCE: EPA Regulations 40 CRF 50; and Commerce Clearing House, Inc., Pollution Control Guide, 1974.

[a]Not to be exceeded more than once per year.
[b]As a guide in devising implementation plans for achieving oxidant standards.
[c]As a guide to be used in assessing implementation plans for achieving the annual maximum 24-hour standard.

always vary in concentration at the same rate, in the same direction, or to the same magnitude as the other members of the class they indicate.

Further, not all the substances in these mixtures have been identified; and a number of the more highly toxic agents which are known are not individually measured, such as some of the small, more easily permeable and damaging particles.

Then, too, conversion or transformation products (for which no standards have been set) are not measured; indications are that some of these may prove considerably more damaging to health.

In addition to these limitations, standards fail to include consideration of the atmospheric interactions, resulting over time in some recognized and perhaps more unrecognized additive and synergistic effects. Excluded as well is any consideration of the health impact on humans of these same pollutants from other environmental pathways such as water and soil.

Aerometric Data

Checking air quality standards for only one or more of the six pollutants thus provides a picture only of a fraction of the total pollution scene. In addition to this, air quality varies with topography from community to community and from neighborhood to neighborhood,

depending upon local stationary and mobile pollutant sources, their intensity, and the duration of their emissions. Air quality varies, too, from day to day, and during the day, and from season to season. It varies also with atmospherics--with temperature, humidity, sunlight, rain, and wind conditions.

Though there are approximately 6,500 monitoring stations across the country, they are still too few and far between, and many are poorly sited (either too near or too distant from emission sources) to furnish a realistic assessment of air quality in many districts. Measuring stations, besides being inadequate in number, are in many cases understaffed and operating with relatively unreliable equipment. And their monitoring instruments are located higher from the ground than the actual breathing zone of adults, and most particularly that of children.

Frequently, in certain areas where monitoring is a recent development, adequate historical aerometric data are unavailable, involving the necessity in calculating risks to chronic low level exposure of resorting to the often crude early estimates of single source emissions or dustfall measurements.

Variations in Susceptibility

A "safe" level tends to imply that a "safe for everyone" level falls below this number, and an "unsafe for everyone" level above this number. This is far from the fact, for marked dose-response variability is the norm. What concerns pediatric interests is the fact that virtually all available data on human toxicology is based on adult studies, and presumably "normal" or "average" adults, and standards have been set on the basis of these studies. In view of children's unique vulnerabilities (as mentioned previously), to calculate a reasonable threshold for various age groups from such adult data would be a difficult exercise at best, since in all probability this would not be a straight-line relationship.

There is a wide spectrum of biological response to pollution exposure among various population groups, and among individuals living at sea level as contrasted with high altitude sites. Adults differ markedly in their reactions, depending upon many physiologic, hereditary, sociocultural factors, and life styles--both outdoors and in. Children, as a subpopulation group, are subject to greater stress from air pollution in at least seven ways different from adults (as covered in Chapter I), but even the pediatric population is a heterogeneous

group. There may possibly be wide variations, for example, between the impact of airborne toxins upon an embryo and/or fetus and adolescent, upon a black child in contrast to a white youngster, or upon a well-nourished as opposed to a malnourished child.

Thus safety margins in all probability fail adequately to assess the relative risks of various pollutants individually and collectively for children and pregnant women in terms of age-, race-, height-, and activity-specific, and other factors.

Besides the difficulties in assessing the effects of interactions between pollutants and their conversion products in the atmosphere above referred to, intrinsic interactions occurring within various bodily systems represent an additional vast unresolved area which still further detracts from a high confidence factor in risk assessment.

Attempts at Quantifying Meaningful Risk Estimates

Despite these recognized deficiences and limitations, two groups (and perhaps more) recently have attempted to put the matter of the health (or disease) aspects of air pollution into some kind of perspective in an effort to determine quantitatively who is at risk to certain concentrations of the air pollutants for which

measurements are available. These are American Public Health Association (APHA) and Environmental Protection Agency (EPA).

Following a brief description of these estimates, an attempt will be made to suggest a more prudent approach to assessment, with reasons why such an assessment is a sounder long-term approach.

APHA Estimates

In this first-cut effort, APHA (1974) chose to identify populations at risk on the basis of SO_2 measurements in areas with 10 thousand population and over, and with two cut-off points: 100 $\underline{u}g/m^3$ and above, and between 65 and 100 $\underline{u}g/m^3$ (the primary standard for SO_2 is 80 $\underline{u}g/m^3$). They acknowledged that this index is not an ideal measure of the underlying forces in polluted air that are most hazardous to health (the acid sulfates being more likely the principal trouble agents). They came up with a figure of 21 million people living in cities--no reference to rural areas--exposed to levels exceeding SO_2 levels of 100 $\underline{u}g/m^3$, and 23 million exposed to concentrations between 65 and 100 $\underline{u}g/m^3$. In the former group were 1.07 million youngsters under 5 years of age, and in the latter approximately 1.2 million under 5.

Obviously, these figures show only a part of the picture of population at risk, since they fail to consider the sulfates, which--as a result of recently disclosed

evidence--would appear to be dangerous at concentrations one-tenth the level set for sulfur oxides (EPA 1974). Though it would seem that SO_2 is presently being controlled quite successfully nationally, sulfate emissions would appear to be increasing; and they're being transported far from their emission sources in rural and suburban steam electric power plants. Also these APHA estimates fail to consider those at risk to other pollutants, as has been mentioned.

Another part of the APHA study dealt not with the standard for an individual pollutant, but with a consideration of various energy inputs from fossil fuels--different ratios of coal, oil, and gas, producing a change in the annual geometric mean of SO_2. They chose as an example for their health model an evaluation of mortality and morbidity in the city of Chicago. Electrical power used by industry and for space heating was measured, but not pollution from automobiles. The "base" case produced an annual geometric mean of 69.4 $\underline{u}g/m^3$ SO_2. They then calculated three specific changes in energy consumption--raising the amount of coal in each from 74.1 $\underline{u}g/m^3$ for Case I to 83.4 $\underline{u}g/m^3$ for Case III.

Of particular interest, is their estimate for the 343,100 children 5 years of age and under in Chicago (according to the 1970 census). A concentration of 83.4

ug/m^3 SO_2, they figured, would result in over 55 thousand excess illnesses per year; these illnesses included both upper and lower respiratory disease. The calculation was made on the basis of a relationship between annual arithmetic averages of SO_2 in the air and the number of acute respiratory illnesses expected per person per year for children 5 years of age or younger, the figures being derived from the New York and Chicago studies from EPA's Community Health and Environmental Surveillance System (CHESS). It has been observed that, with SO_2 above 60 ug/m^3, the incidence of respiratory infection increases proportionately (APHA 1974).

If these findings among the approximately 350 thousand Chicago children under 5 are extrapolated to the 15 million urban children of this same age in the U.S. as of 1975 (assuming that in all these urban areas the concentration of SO_2 was 83.4 ug/m^3), we would have a total of approximately 2.3 million excess respiratory tract illnesses per year. (As will be seen, this figure would appear to be considerably higher than EPA's estimate which follows, except that APHA figures include both upper and lower respiratory disease, while EPA's exclude most upper disorders except for croup.)

EPA Estimates

EPA's latest available information (Takacs and

Shea, 1975) concerning population at risk is calculated on 1973 aerometric readings for those pollutants (with the exception of hydrocarbons) for which national ambient air quality standards have been promulgated in those standard metropolitan statistical areas (SMSA's)--along with their associated suburbs--for which suitable aerometric data were available. (Whether pollution was worse or better in areas where "suitable" data were unavailable is not known.) The coverage was approximately between 50 and 65% of the total U.S. population both for short- and long-term measures* of pollution, with the exception of nitrogen dioxide which was for long-term only. They found that exposure of essentially the entire population surveyed proved to be below primary standards for the following: particulates short-term, sulfur oxides both short- and long-term, and carbon monoxide short-term.

However, a "significant part" of the total population surveyed is exposed to above-standard levels for the following: particulates long-term, oxidants short-term, and nitrogen dioxide long-term. Specifically, for long-term suspended particulates above standard, 31% of the population is at risk; for short-term oxidant levels above standard, 24%.

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*By long-term is meant the annual average concentration; short-term is a 24-hour concentration or less.

They estimated the following numbers of children 0-19 years of age* at risk to levels above the standards for the pollutants named:

Total suspended particulates long-term	14,452,000
Oxidants short-term	22,067,000
Nitrogen dioxide long-term	2,443,000

The constraints of these EPA estimates are obvious, since (as has been mentioned) they fail to include those at risk to other toxic and irritant pollutants in ambient (or indoor) air for which no standards have been set. Nor--very importantly--since present measurement techniques cannot separate out the heterogeneous mix represented by total suspended particulates, there can be no assessment of the relative dangers inherent in different mixes, nor of the submicron particles which penetrate more deeply.

In an 18 August 1974 report for the Federal Power Commission, Finklea and his associates--then with EPA--stated that failure to control SO_4 emissions (as the result of an increase in the use of high sulfur coal) would cause the possibility of otherwise healthy children (age not specified) experiencing from 400 to 900 thousand more common but severe acute respiratory disorders (like croup,

*This is the only age breakdown for children.

acute bronchitis, and pneumonia) each year. Between 1975 and 1980, children would be burdened more frequently with persistent chronic respiratory illnesses if standards are not met, Finklea estimated. He predicted also one to two unnecessary attacks each year for asthmatics. For asthmatic children under 5, this would total approximately 2 million such attacks per year.

As a measure of the uncertainty of all such at-risk estimates, a few months later, in a 27 January, 1975 report, Finklea lowered his estimate of the number of excess respiratory ills to 300 to 500 thousand. How many of these acute conditions might develop into chronic respiratory disorders is an important question (there is suggestive evidence of such an interrelationship, as discussed in Chapter IV).

It is suggested that a more truly realistic estimate of the pediatric population at risk--though it is still very crude and inadequate because it fails to include those at risk to any but the pollutants already mentioned--might be gained by adding to EPA's estimate of those children living in areas where primary standards are exceeded another group of children living in areas with air quality somewhat lower than the present primary standards.

At the present time, the only way it seems possible to accomplish this is to select EPA's class representing

air quality at the first level below the standard, i.e., at 70 to 100% of the primary standard. (It might be sounder to include populations in a still cleaner area, but this is impossible with their data, since the only other EPA air quality classification is from 0 to 75% of the primary standard.)

EPA's estimates for children from 0-19 years living in areas where 75 to 100% of the primary standard obtains are as follows:

TSP long-term	10,631,000
Oxidants short-term	5,096,000
NO_2 long-term	1,697,000

Adding these to the above-standard figure (as shown on page 8), the more conservative estimates as to children at risk are as follows:

TSP long-term	25,983,000
Oxidants short-term	27,163,000
NO_2 long-term	4,140,000

This estimate more than doubles those at risk to TSP long-term and to NO_2 long-term, and increases those at risk to oxidants short-term by a factor of about 5.

In addition in the 75 to 100% category, there are the following susceptibles to two other pollutants:

SO_2 long-term	567,000
CO short-term	679,000

In connection with CO, it is reported (Ayres and Phillips 1975) that, in 1973, none of the largest cities met the standards, with New York City violations (as an example) increasing by 200%.

The totals for these two groups--above primary standards and 75-100% primary standards--are shown in Table 2.

TABLE 2

EPA'S AT-RISK ESTIMATES FOR CHILDREN 0-19 YEARS OF AGE

	Above Primary Standards	75-100% Standards	Total 75-100%+ Standards
TSP long-term	14,452,000	10,631,000	25,083,000
Oxidants short-term	22,067,000	5,096,000	27,163,000
NO_2 long-term	2,443,000	1,697,000	4,140,000
SO_2 long-term	. . . . .	567,000	567,000
CO short-term	. . . . .	679,000	679,000

A Stratified Risk Estimate

Prestigious groups at the national level now strongly advocate the need for environmental health protection (HEW in its recently projected Five-Year Forward Plan for Health for the period 1977-1982, and also the

National Council on Preventive Medicine), and especially for such protection for children (HEW's Secretary for Health). What better place to start than with a consideration of the present standards for air quality?

Because of the tenuous data base--the uncertainties and the unknowns--inherent in the present air pollution monitoring process (as just described), and the tremendous variations in individual susceptibilities to pollution exposure, the band of error is extremely wide in the risk estimates provided by APHA and EPA. Coupled with this are the very significant limitations in the overall perspective of their estimates: their sights have been confined to the effects on only the airways, and no other systems, and to the effects from only six out of a much larger number of pollutants.

Particularly because of the age-specific considerations which render children at greater risk than adults to insults from air pollution, they merit special preventive consideration during their developing years so that they will escape as much as possible the unnecessary stress imposed by dirty air. And because a number of investigators have suggested that present safety margins--especially for certain pollutants--may be inadequate even for adults, it would seem prudent to take a new look at standards from the pediatric point of view. This is of

importance because (as has been stated) standards have been set on the basis of adult toxicologic data. If some standards are proving unsafe for adults, it would seem logical to assume they may be even more unsafe for children in view of their unique vulnerabilities.

Though NAS (1973) has declared what amounts to a "freeze" on present standards--no increase, no decrease, at least for the time present--it is postulated that evidence which has begun to surface and is described subsequently cannot be ignored from a truly preventive point of view, viz.,

(1) the incidence of respiratory disease appears to increase proportionately when sulfur dioxide long-term levels rise above 60 $\mu g/m^3$ (Carnow 1974);

(2) even when SO_2 levels are held at the primary level of 80 $\mu g/m^3$, health effects are observed, apparently primarily due to the acid sulfates, damage from which may develop at much lower--even 10%--of SO_2 levels (Ayres and Phillips, 1975);

(3) lower concentrations of ozone in combination with SO_2 would appear to exert greater bronchoconstrictor effects than either pollutant alone (NAS 1974);

(4) little or no safety margin is associated with the sulfur-dioxide-suspended particulates-fine particulate

sulfate combinations (NAS 1973);

(5) nitrogen sulfates (presently unregulated and for which no standard exists) have produced more deleterious effects than NO_2 (Knelson 1975);

(6) lead at low levels (again, no standard as yet) results in "undue" absorption in many children, adversely affecting brain function and producing neuropsychological defects (see Chapter IV);

(7) CO in low dosage may also be affecting children's CNS.

Furthermore, as additional research is processed, complications now suspected may be confirmed, indicating that a more conservative approach to pollution control is eminently sound for the long-term health of children. Among these possibilities are the following:

(1) more additive and syneristic effects both with the primary agents and their degradation products as well as within the host may be identified;

(2) the bronchitic child may indeed prove to be father to the bronchitic man--i.e., the tendency toward chronicity developing early;

(3) other systems and subsystems besides the respiratory and central nervous systems may be considerably impaired;

(4) undesirable effects on general growth and

development and further evidence with respect to an adverse impact on mental health may be demonstrated (see Chapter IV); and

(5) possible association of unregulated trace metals, pesticides, plastics, etc., with carcinogenic, mutagenic, and teratogenic sequelae may be established--see Chapter IV.

For these reasons and others, it seems the part of wisdom to consider all children from 0-14 years at risk to airborne pollutants, with some at higher risk than others. This more prudent estimate is scaled as follows as shown in Table 3.

TABLE 3

STRATIFIED RISK ESTIMATE

At risk: all children under 14 years		60 million
At high risk: all under 5 years		20 million
At very-high risk: urban under 5 years		15 million
At very-high risk: especially vulnerable groups, including:		
blacks under 14 years	6	million
poor under 14 years	7.8	million
asthmatics under 14 years	3	million
lead-endangered under 6 years	1	million
pregnant women	3	million

It will be noted that the 15-19-year group is

excluded in this estimate because, in a certain percentage of cases at least, cigarette smoking may exercise a stronger adverse influence than community air. Among the specially vulnerable groups, the black and the poor are included because, in general, they suffer greater residential exposure to air pollution, and have a lower resistance to disease (Brodine 1973). Asthmatics are included because of their heightened sensitivity to respiratory tract irritants. Those already exposed to leaded paint as a function of their housing are also included, as are pregnant women because of the risk to embryos and fetuses.

CHAPTER III

CONTAMINATED INDOOR AIR ALSO A RISK

On the average people are said to spend 80% of their time indoors, and those who may be at higher risk to airborne pollutants--infants, young children, and pregnant women--may spend more. Though the indoor environment has frequently been ignored as a significant source of exposure to air pollutants, indoor air quality may in fact prove a prime determinant of an individual's total pollution exposure.

Very few data have been gathered on the presence, concentration, and generation of pollutants in indoor environments, and on the penetration of pollutants from outdoor environments into buildings. Obviously contaminated air doesn't stop at the door of a home--though penetration varies with the season of the year, the type of building design, construction, insulation, ventilation, etc. In some studies based on limited carbon monoxide and particulate data (Yocum 1971), pollution levels inside houses appear to be slightly higher than those inside offices and public buildings when similar outdoor concentrations prevailed.

As of 1972, the best available estimate of indoor concentrations of particulates and non-reactive gases, according to the U.S. Environmental Protection Agency (EPA), was that, in general, they equalled outdoor concentrations. It has been recognized for some time that carbon monoxide, sulfur dioxide, and particulates can be generated by interior activities involving combustion, i.e., smoking, cooking, and heating (Figure 2). New evidence points to the fact that, under certain circumstances, indoor concentrations may actually exceed outdoor concentrations for certain pollutants.

In July 1974, three NERC scientists (Bridbord et al. 1975) observed that there are a number of documented examples of levels of indoor pollutants greatly exceeding those levels which commonly occur in the outdoor environment. Among these instances, they cite the airborne buildup of polynuclear aromatics and cadmium from cigarette smoke, lead from burning candles, and vinyl chloride from use of aerosols containing this substance as a propellant.

Principal Indoor Offenders

Tobacco Smoke

Perhaps the single, most potent source of chronic irritation in the home (and car) comes from tobacco smoke in any form: its dangers to fetuses particularly have been

Fig. 2. Contaminated indoor air from many sources

52

51

quite thoroughly documented (HEW 1973).

Effects on Fetus

Smoking during pregnancy--as a result of one or more of possibly half a dozen different mechanisms--affects the unborn child most unfavorably in at least two critical ways.

The first effect concerns birth weight: HEW states that every investigator who has examined the relationship has confirmed that infants of women who smoke during pregnancy have a lower average birth weight than the infants of women who do not and there is much evidence to indicate that it is the cigarette smoking during pregnancy which causes this reduction in infant birth weight. Low birth weight children--as is well known--are at a distinct disadvantage healthwise.

The second effect concerns mortality. Where several investigators have demonstrated that the fetal and neonatal mortality rate is significantly higher for the infants of smokers as compared with those of nonsmokers, other investigators have not found this to be the case.

Various estimates of increased fetal and infant mortality range from 28 to 60% and higher among smoking mothers.

As for the teratogenic effect of tobacco smoking,

no conclusion can be drawn at this time, since studies of the association between maternal cigarette smoking and congenital malformations have produced conflicting results.

Infants and Young Children

Besides the fetus, the infant may also be adversely affected by mothers and fathers who smoke. Several recent studies indicate that sidestream smoke from the burning of cigarettes contains compounds in higher quantities than in the mainstream smoke inhaled by the smoker (Steinfeld 1975). Besides carbon monoxide, these compounds include tars, nocotine, and nitrous oxides.

As a result of its 5-year study of over 2 thousand children, a British team (Colley et al. 1974) reported that exposure to cigarette smoke generated when both parents smoked doubled the risk for the infant of an attack of pneumonia or bronchitis. The risk was lowest where both parents were nonsmokers, highest where both smoked, and lay between these two levels where only one parent smoked. In contrast, between 1 and 5 years of age, they observed no important effect of passive smoking in increasing the risk of pneumonia and bronchitis. And, of the long-term impact of such illness among those who survive, the authors say this:

> The evidence points to some damage to the respiratory tract as indicated by an increased prevalence of chest symptoms and deficits in ventilatory function found in later childhood . . . If by the age of 20, such long-term effects are found, these could persist into middle and late adult life, and contribute to the evolution of chronic respiratory disease.

EPA, in four of its community studies involving children, reported mostly positive but also negative findings with respect to the relationship between cigarette smoking in the home and the incidence of acute lower respiratory disease. In the Salt Lake Basin area (Nelson et al. 1974) among some [illegible] thousand preschool and elementary school youngsters, "significant"* increases in acute bronchitis and pneumonia occurred in those preschoolers most heavily exposed to home pollution from sidestream cigarette smoke; however, socioeconomic factors were not separated out. In 627 familes of relatively high socioeconomic status in Chicago and northwest Indiana (Finklea et al. 1974), a "definite increase" ($p < 0.05$) in the risk of acute lower respiratory illness in nursery school children was associated with cigarette smoking in the home. Likewise, in their 1970-1971 New York investigation (Love et al. 1974), in three communites "reasonably similar in general socioeconomic status," cigarette smoking in the home significantly ($p < 0.01$) increased acute lower respiratory frequency among approximately

*degree of significance not supplied.

2,500 school and preschool children. However, in 1972, in four other New York communities (Hammer et al. 1974), no difference could be detected in lower respiratory disease morbidity among close to 8 thousand black and white socioeconomically similar children (aged 1-12 years) of smoking and nonsmoking parents.

A few years previously, two surveys involving almost 2 thousand families by a Wayne State University team (Cameron et al. 1969) disclosed that smokers' children under 15 years of age were sick more frequently (primarily respiratory illness) than nonsmokers' children. And there was some evidence that, among the children exposed to smoke, those who were ill were exposed more. (No reference to socioeconomic status.)

Smoking Children

As for youngsters who themselves smoke, The American Academy of Pediatrics (AAP 1970) cites a number of studies pointing to evidence that they have more respiratory symptoms, cough, phlegm, breathlessness, wheezing, and colds than the abstainers. Also boys who smoke have a higher incidence of traumatic injuries and girls a higher incidence of urinary infections, which may or may not be a result of their habit or addiction to tobacco.

Aerosol Sprays

One of the activities of people which strongly affects indoor pollution is the widespread use of aerosol spray products--literally hundreds of items available at drug, hardware, department stores, and supermarkets--for everything from air fresheners to waxes, including toiletries, cosmetics, cleansers, spot removers, germicides, fungicides, insecticides, and paints. As Carnow (1973) put it,

> Some women may create an alarming indoor pollution crisis every morning in their bathrooms by using a hair spray, underarm spray, feminine hygiene spray, or foot spray. The clouds of particles emitted from these sprays may enter the lungs. There may be no sign of damage for 10 to 20 years.

Some women create equally alarming pollution crises every morning in the kitchen, too, and at other times of the day and night in other parts of the house as they wield spray cans in the pursuit of their household chores (or hobby work)--in cleaning, polishing, waxing, lacquering, mothproofing, fire-retarding, and the like.

These sources of indoor air pollutants from aerosol products may, in the view of the National Environmental Research Center (NERC), "build up to concentrations of potential public health concern." Hester and his associates (1974) have reported studies of instantaneous freon 11 and 12 concentrations* in the indoor

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*Freon is duPont's brand name for fluorinated hydrocarbon.

environment taken at random which confirm the presence of undesirable quantities of these pollutants in the indoor air: 500 ppb as contrasted with simultaneous outdoor concentrations of 1 ppb or less.

In some indoor environments, the concentration of halogenated hydrocarbons--as for example, perchloroethylene and trichloroethylene (a structural analog to vinyl chloride, a known carcinogen and liver toxin, and possibly also a mutagen)--exceeds established exposure limits for industry workers. This threshold limit for these two halogentaed hydrocarbons is 100 ppm.

Another example of such indoor contamination is a study made by duPont Laboratories (Center for Science in the Public Interest 1973). The concentration of Freon propellant in the air in front of the faces of users of hair spray and deodorant in a room ventilated with an exhaust fan was as high as 370 and 360 ppm. Such concentrations are considered a "cause of concern" for several reasons, including the danger to eyes (propellants can cause corneal irritation); to the skin (they can cause freeze burns); to the heart (they can cause cardiac arrest); and possibly to the lungs (perhaps producing enlarged lymph nodes and thesaurosis in some cases); and to increased abortion, mutations, cancer, etc.

Besides the fluorinated or halogenated hydrocarbons,

there are other undesirable propellants used in aerosol cans: propane, butane, isobutane, carbon dioxide, nitrous oxide, and methylane chloride. But, in addition to the pervasive, intensive action of the propellant, which--as a fine mist--is easily inhaled, two other elements are usually contained in aerosol cans: a principal ingredient and a solvent, either or both of which can also exert deleterious action. Among such are the two halogenated hydrocarbons mentioned above, carbon tetrachloride, and numerous anesthetic agents as well as vinyl chloride.

As increasing efforts are made to insulate buildings more efficiently to conserve energy, the problem of indoor pollution as a result of the widespread, frequent, and sequential use of aerosol products can easily worsen. As yet, the long-term effects of low concentration of the ingredients of many aerosol products have not yet been adequately investigated; it is obviously an area urgently requiring research.

For children especially, in addition to possible irritation and toxicity from the inhalation of aerosol spray products, there are two other serious dangers: from fire and explosion. The high flammability of many of the ingredients--such as alcohol, propane, etc.--is well established, as is also the explosive nature of the gases

contained in the cans.

Heating, Ventilating, and Cooking Equipment

Faulty heating equipment--oil- or coal-burning systems particularly--can generate considerable pollution from carbon monoxide, sulfur dioxide, and particulates. Gas furnaces or heaters are usually not significant sources of indoor carbon monoxide; on the other hand, gas ranges apparently are. So, too, are attached garages.

There are several pediatric implications with respect to excess carbon monoxide. In the first place, the developing fetus is unusally sensitive to insufficient oxygen. The newborn infant is also. If chronic carbon monoxide intoxication is a substantial risk factor in atherogenesis, a relatively high percentage of children may be involved. In a nationwide study of a "large group" of children (number not specified) conducted by the Bureau of Community Environmental Management (1974), almost a quarter had carboxyhemoglobin levels of 3% or more (which levels have been implicated in heart illness). Suspected cause: defective home heaters and stoves. Carbon monoxide has a number of other health implications, including its deleterious effect on the central nervous system, mental acuity, concentration, vigilance, etc.

Apparently it is not unrealistic to fault various

combustion appliances now in use, for--in a check of 372 homes with over 1 thousand such units--25% were found to be discharging carbon monoxide into the room atmosphere--and 25% of these in the range of 200 ppm and over by volume (EPA standard for 8-hour exposure is 9 ppm; for 1-hour exposure 35 ppm). In the carbon monoxide-positive homes, 40% of the inhabitants gave clinical histories of carbon monoxide anoxia. Yates (1967) concludes from his limited data that continued exposure to subacute concentration of CO in homes could be a predisposing cause of some undiagnosed illnesses.

As for the effect of nitrogen dioxide from gas stoves or space heaters on respiratory tract illness, EPA--in its community studies in New York--found no significant difference (Hammer et al. 1974), but in another attributed a "modest excess" in frequency of acute lower respiratory illness in the use of gas (Love et al. 1974).

WHO (1972a) pointed out that not only can smoke from inefficient heating appliances irritate larynx and bronchi, but gas piped to houses for heating and cooking is a very frequent cause of accidental poisoning and sometimes death due to carbon monoxide inhalation.

Gas stoves can also emit nitrogen dioxide in concentrations which can prove irritating. In fact, EPA, in a "preliminary" study (Jennings 1974), found that

improperly vented gas stoves can result in a nitrogen dioxide concentration 20 times the Federal air quality limit of 0.5 ppm. Travelers Research Corporation (Susman 1975) found somewhat similar results for excess nitrogen dioxide concentration and for carbon monoxide as well.

Space heaters also frequently produce carbon monoxide and nitrogen compounds. Wood-burning fireplaces, too, can generate irritating and/or dangerous fumes and smoke.

Housedust

Particles present indoors are either resuspended or kept in suspension by people activity. During the day, they are distributed rather uniformly from floor to ceiling; at night, however, they tend to settle and become concentrated near the floor. High particle counts from cleaning and dusting persist over a period of at least several hours.

Lead in housedust can prove a serious pediatric problem (described in further detail in Chapter IV). This can arise either from lead-based paint used indoors or out, or the lead from car exhausts permeating the indoor atmosphere. From either source it is especially deleterious to young children. Dust analyses of smears from floors, walls, and windowsills in low-income inner city dwellings have shown a median concentration of lead five

times as high as in suburban homes (Vostal et al. 1974). But lead-laden housedust is not confined solely to low-income homes, several studies indicating that, in middle-class residences in urban areas, lead levels in housedust are roughly double those in suburban middle-class residences (EPA 1972).

Asbestos dust is hazardous also because of its potential in causing mesothelioma, lung cancer, and other debilitating lung damage. There is no solid data as to the threshold concentration for continuous exposure to ambient concentrations of asbestos, but there are many asbestos-containing household items--everything from talcum powder (some of which contain a higher percentage of respirable fibers) to ironing board covers, drapes, filters of various sorts, clothing, pads and covers, stove mats; and also to a growing list of building materials, including floor tiles, and electrical insulation.

In connection with woolen coats with an interweaving of asbestos, Selikoff (1971) has said that if he were a woman with a coat like that, he would bury it. He explained that brushing or manipulating the coat raised asbestos levels in the air to 10 thousand times the normal concentration.

Preventive Measures

Fortunately opportunities exist to upgrade indoor air quality. There are steps which individuals can take to reduce pollutant concentration in homes, schools, day care centers, and wherever else children are at work or play; also in private automobiles or school buses in which children are transported. The exception of course, is the location of residences and schools in terms of propinquity to noxious outdoor emissions from industrial, automotive, and other sources. This is not always subject to our control for a variety of reasons (though planning for new homes or schools should take this into consideration). Among important measures that can be embraced are the following:

The first and one of the most obvious preventive steps concerns elimination of the use of tobacco products. The urgent need for action in this direction should require no further elaboration. Yet in 1970, approximately one-third of American women of childbearing age were cigarette smokers. The percentage of U.S. women who smoked throughout pregnancy is not definitely known, but is presumably lower, probably in the neighborhood of 20 to 25%. However, smoking is increasing among women, and particularly among teenagers. There are said to be 500 thousand more teenage girl smokers now than in 1969, with a heavy percentage of

these smoking at least a pack a day (Brody 1976).

If smoking adults fail to break the habit for the sake of their own health, can they be persuaded to curb their addiction for their children's benefit?

Another preventive measure deals with aerosols, the use of which has increased exponentially in the past few years. Since it appears so obvious to so many that the risks from the propellants and active and solvent ingredients in aerosols so far outweigh the advantages, this pollution source can easily be controlled by interdicting all such products. A number of alternatives are available that are both safer and cheaper. Examples are manually operated sprayers, soaps, powders, liquids, sticks, pads, creams and roll-ons.

Contraindicated also is the use of asbestos- and lead-containing products, which are legion, as has been elucidated elsewhere. Homes should be well maintained in terms of promptly cleaning up dried plaster and flaking paint which can contribute to lead-laden dust. In extensive patching jobs or in duct work around the home, care should be taken to avoid commercial spackling and dry wall taping compounds which contain asbestos. When mixed or sanded, these compounds can cause dangerous fibers to be spread as dust around the area.

Likewise, poorly vented heating and cooking equipment

should be strictly avoided, and--when discovered--quickly corrected. It isn't always easy to detect carbon monoxide emissions, but telltale signs--besides odor--are smoke, sooty deposits around registers, exhaust discharges, and vent pipe joints.

Air-conditioning can improve air quality indoors to some extent. However, whether air conditioners can filter out the small, more dangerous particles is a question. The effectiveness of the equipment is entirely dependent on the type, and on the degree of cleanliness with which it is maintained. This applies to filters or cleaners of whatever type. As an example, dirty humidifiers can recirculate troublesome agents, and vacuum cleaners can do likewise.

The family car should be considered part of the indoor environment. So should school buses--where many children spend as much as 200 hours or more each year. And, in addition, skating rinks. In all of these places, carbon monoxide poisoning has occurred as a result of excess tobacco smoke, faulty exhaust systems, or in arenas where ice-resurfacing machines were used.

CHAPTER IV

HOW CHILDREN ARE (AND MAY BE) AFFECTED BY AIRBORNE IRRITANTS AND TOXINS

A simplistic way of stating how air pollution affects children is to say that it's bad for their health. It's bad in a number of ways--some recognized, perhaps more unrecognized--with each individual as a biological organism, reacting according to his unique degree of resistance and adaptive capacity, and his particular stage of growth and development. The smaller the fetus or child, the more delicate and defenseless the organism--including the genetically handicapped, the malnourished, the poor, the diseased--the greater the risk from any additional physiologic (or psychologic) burden such as that imposed by noxious airborne solids, gases, or liquids.

There is a wide spectrum of psychobiological response to pollution exposure; there is also a wide spectrum of type of pollution, depending upon emission sources and types, upon weather, topography, and atmospheric conditions (as mentioned in Chapter II). And at different levels of pollution, someone becomes ill, and someone's life is shortened; but many of the effects are

unidentified. The more protracted the exposure (both indoors and out), the more toxic the mix of chemicals and metals, the weaker the host, and the closer to emission sources and/or to concentrations of dangerous pollutants--the more serious the insult and ensuing damage.

Pollution Vulnerability Index

Thus a number of factors--intrinsic and extrinsic--affect a child's reaction to air pollution. Besides the intrinsic factors (the special vulnerabilities of embryos, fetuses, infants, and children which make them more susceptible than adults) mentioned in Chapter I--tidal volume respiratory illnesses, location of breathing zone, play activities and habits, and stage of growth and development of various systems and subsystems--each child reacts according to his individual state of health, genetic inheritance, and nature and degree of immunocompetence.

The extrinsic factors vary according to the propinquity and intensity of effluent generation from industrial, power, incineration, and transportation sources. Also, the atmospherics and the physical and chemical synergisms and additives operative in the particular locus in which his parents have placed him. Again, with any potentially harmful occupational exposure of father or mother. Combined, these extrinsic and intrinsic factors comprise what might be described as each child's individual "pollution-vulnerability

index" (Table 4).

TABLE 4

POLLUTION VULNERABILITY INDEX

Intrinsic Factors	Extrinsic Factors
Genetic inheritance	Propinquity to emission sources
Age	Concentration of emissions
State of health	Topography
Breathing zone area	Climate
Respiratory problems	Weather
Play activities	Atmospherics
	Parental occupation

The acute reactions to air pollution are obviously more readily recognized; it's the exposure to pollution over the long-term which is more difficult to assess. It is an examination of the effects of this chronic exposure over time which is the primary focus of this exercise, as has been mentioned.

Non-Allergic Airways Disease

The airways (respiratory) system would appear to be most subject to irritation and/or toxicity from inhaled polluted air, and more is known concerning the impact of ambient air on lungs than on most other organs. But many

systems besides the respiratory are affected by airborne pollutants, and they will be discussed below.

It would seem most logical, therefore, to commence this exploration with a consideration of air pollution and the respiratory tract, since--among the acute conditions of childhood--illnesses of this tract account for 50 to 70% of all pediatric morbidity, and for approximately 30% of all deaths in children under 15 years of age. Also, more than 60% of the total school days lost to illness are due to respiratory disease.

With respect to air pollution and the respiratory system, the following findings would appear to be quite well documented, not only from research in the U.S., but in England, Japan, and other countries as well:

(1) Pollution affects mortality from respiratory disease and other conditions in the first year of life;

(2) Pollution depresses lung function;

(3) Pollution increases the incidence of acute lower (and perhaps upper) respiratory tract illness in children (and possibly their families as well);

(4) Pollutants may impair the multiple defense mechanisms of the respiratory tract, increasing susceptibility to bacterial and viral infections;

(5) Pollutants may contribute to the development of chronic bronchitis in later life; and

(6) Pollution aggravates asthmatic conditions.

Mortality

On the mortality issue occurring as a result of acute air pollution episodes, the concenses would seem to be that air pollution in the first year of life does have an effect on mortality from respiratory disease and other conditions (Holland 1974). However, Greenberg, from his studies of the 1963 New York City air pollution episode, does not concur (NAS 1975). On the other hand, Lave and Seskin (1972), in their statistical analyses, point to an association of air pollution with neonatal mortality.

Pulmonary Function

Simple tests measuring pulmonary function can be used as an index of disease, since they can detect impairment earlier than by either clinical or radiographic examination. Decreases in ventilatory function occur with aging, with chronic obstructive lung disease, and among smokers. In children, it is generally agreed that there is a diagnostic value in determining the vital capacity of the lungs, i.e., that the status of ventilatory function is a measure of the health of the respiratory tract. It is held by many also that the significance of decrements in ventilatory performance early in life is that they may represent a risk factor for later respiratory disorders.

Various factors besides the health of the respiratory tract influence response to ventilatory function tests. Among these are height, age, sex, race, socioeconomic status, and weather. A number of researchers have measured the impact of air pollution on children's ventilatory function. Most all studies--primarily those first done in England (Holland et al. 1969 and Lunn et al. 1967, 1970) and Japan (Kagawa et al. 1974, Toyama 1964, and Watanabe et al. 1964), and in other countries as well--have demonstrated "quite clearly" that ventilatory function, as measured at various ages, is influenced by three factors: area of residence (meaning areas of high as contrasted with low levels of pollution), social class, and a history of pneumonia or bronchitis in the first four years of life (Holland 1974). However, this may not be the case for the newborn infant, according to a recent unpublished study.*

While air in Great Britain may be dirtier than U.S. air, particularly with respect to smoke and sulfur dioxide, this is not always the case (NAS 1974). And in two Japanese studies, ventilatory function responses appear

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*In a group of about 2,500 families in which Holland and Wysocki (1974) measured ventilatory function of the baby within four weeks of birth, no influence was found with respect to area of residence (as an index of pollution), or to any of the factors that could influence the mother during her pregnancy.

to occur at relatively low pollution concentrations of sulfur dioxide and particulates (NAS 1975).

In some studies, the effects appeared to be reversible when air quality improved: this is apparently the view of the Japanese investigators. Others, however, are inclined to accept the difference as a permanent character. The effect of prolonged exposure is at present unknown.

What about American children? How do their lungs perform under air quality conditions as they prevailed in the late 1960's and 1970's in various geographic areas in the U.S.? Appendix A provides considerable details on these.

EPA studies have examined ventilatory function in elementary school children living in neighborhoods of high and low air pollution (of various mixes) in a number of different parts of the country--in Chattanooga, Tennessee, Cincinnati, Ohio, New York City, Charlotte, North Carolina, and Birmingham, Alabama (and possibly other places as well). Their teams of investigators are confident that they, too, have demonstrated that air pollution decreases ventilatory function, as will be explained, but their work has been heavily criticized, perhaps overcriticized (criticisms of studies, where found, have also been included in Appendix A).

First, the Chattanooga studies (Shy 1970 and 1973) on almost 1,000 second grade children in 12 schools in four areas (two polluted--mostly by NO_2--and two controls). Ventilatory performance (FEV. 0.75*) was significantly ($p < .05$) lower in high exposure areas, but adversely affected only when the NO_2 threshold was exceeded. This study has been faulted on the grounds of unreliability of monitoring measurements, simultaneous presence of other pollutants, and somewhat less than a straight-line dose-response relationship (Griffin 1974). On the other hand, Wehrle (Chairman of AAP's Committee on Environmental Hazards), in his summary presented to AMA's 1974 "Air Pollution Medical Research Conference," stated as follows:

> Ventilatory performance of second-grade school children residing in the area with high NO2 concentrations was significantly decreased when compared with children in control areas nearby. No adverse effect was found among children from the area with increased levels of suspended particulate matter.

In EPA's Cincinnati study (Shy et al 1973 and 1974), approximately 400 second-grade children in six neighborhoods differing in socioeconomic level, race, and pollution exposure were measured for ventilatory performance. Moderate levels of air pollution exposure had a definite effect on ventilatory performance, as did

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*A three-quarter second forced expiratory volume measurement.

socioeconomic status and weather (the cold winter months when pollution was highest). Race was also a factor: pollution exposure had no consistent area effect on the performance of black children. Shy and his team attributed most of the pollution effect* to suspended sulfates, but observed that the relative effects of total suspended particulates, suspended sulfates, and suspended nitrates need further elucidation, particularly in combination of the latter two.

Over 2,300 white children aged 5 to 13 years took part in EPA's investigation in the New York City metropolitan area (Shy et al. 1974) of three different pollution exposures. Older white children (aged 9 to 13 years) from a relatively unpolluted suburban fringe community on Long Island were found to have significantly better ventilatory function than children from two more polluted urban communities. This difference, it is pointed out, existed in the face of recent improvements in air quality.

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*Sulfur dioxide levels were low: the 7-month arithmetic mean was less than 52 ug/m^3 in all sectors (primary standard= 80 ug/m^3). Levels of tsp were 76 to 131 ug/m^3 (primary standard= 75 ug/m^3) in polluted sectors and 61 to 92 ug/m^3 (no standard) compared to 8.3 ug/m^3 in low-exposure neighborhoods of the whites; average suspended nitrate level was 3.1 ug/m^3 compared to 2.4 ug/m^3 in clean white sectors.

Older children exposed to higher levels* for more than eight years showed significant decreases in ventilatory function, but the 5- to 8-year-olds did not. The investigators are of the opinion that the impaired function induced by long exposure to high pollution levels may persist even as air quality standards are achieved.

Both the Cincinnati and New York City studies are downgraded for a number of reasons--primarily inconsistencies in the data and relatively small observed differences (Higgins and Ferris 1974). EPA, in acknowledging that the differences in the two studies--plus that in Chattanooga--are slight (varying from 2 to 17%), believes that, because impairment was observed at an early age, the findings are rendered all the more significant in terms of disease prevention (Shy et al. 1973). On this point, (Elliott Goldstein, NAS 1973) concurs, expressing this view:

> . . . the arguments concerning the medical importance of a slight albeit significant decrease in certain relatively crude measurements of ventilatory function seems to be valid.

One of the objectives in another EPA survey**

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*Annual average SO_2 levels estimated at 131 to 435 $\mu g/m^3$, accompanied by levels of suspended particulates estimated at 75 to 200 $\mu g/m^3$, and suspended sulfates estimated at 5 to 25 $\mu g/m^3$.

**This was possible because the two cities had low levels of other pollutants.

involving 8,000 white and black elementary school children in Charlotte, North Carolina (a cleaner area) and Birmingham, Alabama (a dirtier area) was to check the impact of suspended particulate matter with two fractions of particulate interest: suspended sulfates and respirable particulates of a diameter about 3.5u or less.* In all eight age-, sex-, and race-specific subgroups, mean age- and height-adjusted FEV 0.75 readings were consistently lowest in the more polluted city (Birmingham). The principal investigator (Chapman et al. 1974) felt that this finding "strongly indicated" that exposure to particulate pollution had exerted a deleterious effect on the FEV 0.75 of children in Birmingham.

Their results, they believed, were most consistent with two alternate hypotheses: that exposure to respirable suspended particulates (including suspended sulfates) from the beginning of life onward promotes impairments in FEV 0.75 in later childhood; and that suspended sulfate exposure for the past several years promotes such impairments.

Though NAS (1975) criticized this work for "a number of experimental shortcomings," the conclusion was expressed that (as is true of other CHESS studies), the

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*The first of a 5-year series of assessments of children's ventilatory function in these two cities.

"major positive finding is the consistency of an effect of air pollution."

Other Studies

Other investigators in the U.S. not associated with EPA have also observed the action of air pollutants on pulmonary (and other) functions of American youngsters, notably the following two:

A 1966-1967 examination (Ferris 1970) of first- and second-graders from seven schools in Berlin, New Hampshire, showed small but statistically significant differences in pulmonary function, particularly peak expiratory flow rate, that suggested an effect of air pollution, apparently mostly due to particulates (NAS 1975).

In 1970 and again in 1973, biologists at the University of Akron compared cardiopulmonary functions in 42 high school students from an urban industrialized area with 50 from a rural area (Mostardi and Leonard 1974). Four different measures were employed. The conclusion was that air pollutants should be considered "important contributory factors" in the impairment of cardiopulmonary functions. In reviewing this study, NAS (1975) observed that it tends to support an association between a decrement in childhood pulmonary function with air pollution slightly above the current U.S. standards for total

suspended particulates and sulfur dioxide.

In a recent report investigating the action of air pollution of either oxidant or reducing type and weather on lung function in exercising children and adolescents, Lebowitz et al. (1974) found significant decrements (6.1%) on days of high--as compared with low pollution--temperature days (0.4%). The researchers noted that exercise per se does not decrease lung function, nor do such factors as ethnic group, social status, and respiratory history appear to affect it. They state that their results confirm a recent study among sixth-graders in Los Angeles, in which reduction of lung function after exercise was correlated with atmospheric ozone concentration.

Though certain investigators (like Biersteker and Van Leeuwen 1970) have reported negative results, it seems quite generally accepted that pollution of various mixes, particularly over time, produces a decrement in children's pulmonary function.

Acute Respiratory Illness

Let us consider now the recent findings in the U.S. concerning air pollution and its relationship to the incidence and prevalence of lower and sometimes upper respiratory tract infections in children (and on occasion in their families as well).

Before highlighting the findings of the relevant EPA Community Health Environment and Surveillance System (CHESS) studies, brief reference should be made to British (Colley 1971; Colley and Reid 1970; Douglas and Waller 1966; and Lunn et al. 1967) and also to Japanese (Toyama 1964) research--most using sulfur dioxide and particulates as the index of pollution. These have demonstrated that there is more respiratory illness in children living in polluted areas than in those breathing cleaner air. Indeed, both frequency and severity of lower respiratory infections at all ages are related to the degree of exposure.

EPA has attempted to overcome what they regard as deficiencies in the British and Japanese studies, which--while yielding remarkably consistent results--failed to achieve these two important goals: (1) separation of the effects of sulfur dioxide from those of suspended particulates, and (2) relating the length of exposure to excesses in acute respiratory morbidity. Other limitations of the overseas investigators included rudimentary environmental monitoring, and inability to consider important socio-cultural covariates.

Effects of Sulfur Oxides

Accordingly, EPA projects were designed in an attempt to fill these gaps and overcome these deficiencies. How did they approach this? First by retrospective

surveys* of the frequency of acute lower respiratory disease among approximately 20 thousand children (aged 1 to 12) and their families in the following areas (each with a somewhat different pollution situation): Salt Lake Basin communities (characterized by high sulfur dioxide levels, high sulfates, and low total suspended particulates); in five Rocky Mountain communities (high sulfur dioxide, high metals, and low total suspended particulates). And second, by two prospective surveys** of acute respiratory disease in volunteer families in Chicago and New York City--both of these metropolitan areas characterized by high sulfur dioxide and high total suspended particulates.

In the two retrospective studies in the western areas--Salt Lake Basin and Rocky Mountain communities--the EPA team believe their data "clearly linked" excessive, acute lower respiratory tract infections with communities heavily polluted with sulfur dioxide and suspended sulfates--this effect being independent of socioecnonomic status and cigarette smoking. Pollution exposure

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*An epidemiologic method which looks back into health and medical histories to identify causes of disease.

**An epidemiologic method which examines cohorts of exposed vs. unexposed individuals over time to define frequency of subsequent disease in relation to a wide dose-range of agent(s).

of three years or longer was an important component in increased risk (Finklea et al. 1974).

Summarizing further, EPA observed these four consistent findings:

(1) For all combinations of disease and numbers of illness episodes, no significant association between total respiratory disease and pollution was found for children whose parents had been residents of their communities for less than three years.

(2) For single and repeated episodes of croup and repeated episodes of any lower respiratory disease, families of children who had lived three or more years in high exposure communities reported more illness across all ages of children from 0-12 years than did their counterparts in less polluted communities.

(3) For single or repeated illness episodes and for all residence durations, there were no association of pollution exposure with pneumonia or number of hospitalizations for total lower respiratory disease.

(4) Socioeconomic status was not a factor in illness rate, but age was: the young were affected more frequently.

Thus excess respiratory disease--in EPA's view--may reasonably be associated with community exposure to approximately 95 $\underline{u}g/m^3$ sulfur dioxide (standard= 80 $\underline{u}g/m^3$) and

15 ug/m^3 suspended sulfates (no standard); also there is no evidence that elevated levels of total particulate matter are required to produce the adverse effect.

NAS (1975) denigrated both these studies on several grounds (the Rocky Mountain findings were found "not as impressive"), but of the Salt Lake study it was stated that in general, the data do appear to substantiate an increase in lower respiratory disease* in children exposed for a few years to sulfur dioxide at 90-100 ug/m^3 and suspended sulfates at 15 ug/m^3 in the presence of total suspended particles at concentrations below the air quality standard.

As a result of their two prospective studies of acute respiratory disease in New York City and Chicago families--involving approximately 6 thousand individuals--EPA reported a consistent excess acute respiratory disease rate among family members living in more polluted neighborhoods. Socioeconomic status was a significant factor in the incidence of upper but not lower respiratory disease.

They (Finklea et al. 1974) "conservatively" estimated that exposure to 210 ug/m^3 sulfur dioxide with 104 ug/m^3

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*This increase in the Salt Lake area amounted to 40 to 50% after exposure to elevated levels of sulfur dioxide (91 ug/m^3) and suspended particle sulfates (15 ug/m^3) lasting over two years.

total suspended particulates, and approximately 16 ug/m^3 suspended sulfates was associated with a 5 to 20% excess of acute respiratory infection in various family members.

Smoking was a significant determinant in the development of lower tract illness as a result of the initial infection. Parental smoking also was a significant factor in the development of lower tract illness among nonsmoking young members of their households.

The Chicago study also provided evidence, EPA stated, of increased susceptibility to epidemic A_2 Hong Kong influenza among otherwise healthy families exposed during the previous three years to atmospheric levles of 105 to 119 ug/m^3 sulfur dioxide, 151 to 159 ug/m^3 total suspended particulates, and 14 ug/m^3 suspended sulfates.

In both the New York City and Chicago studies, the investigators are of the view that even more recent and substantially lower air pollution levels contributed to increased respiratory illness. In New York City, this involved an annual average sulfur dioxide level of 51 to 63 ug/m^3 and annual average suspended sulfate levels of 13 to 41 ug/m^3.

In sum, Finklea (1973) stated:

> Families living in more polluted urban areas report excesses in common respiratory illness which were roughly comparable to the excesses in acute respiratory illnesses observed nationally during years of epidemic influenza like 1965-1966 and 1968-1969.

Of these family studies, NAS (1975) believes that: they tend to add further weight to the reported association of childhood lower respiratory illnesses with concentrations of sulfur dioxide and total suspended particles above the current U.S. air quality standards.

Still another--later--project of EPA is their 1972 retrospective survey in four New York communities representing different levels of exposure to suspended sulfates, particulates, and sulfur dioxide. Hammer et al. (1974) reported a significantly higher rate of any lower respiratory disease with croup, bronchitis, and other chest conditions among non-asthmatic black and white children aged 1 to 12 years living in the higher pollution exposure communities. In fact, his study showed that acute bronchitis was reported about 20% more frequently among the exposed children.

Hammer's "best judgment" estimate of the levels required to produce effects would be exposure to average annual levels of approximately 175 to 200 $\underline{u}g/m^3$ of sulfur dioxide, 85 to 110 $\underline{u}g/m^3$ of total suspended particulates, and 13 to 14 $\underline{u}g/m^3$ of suspended sulfates. Of these data, NAS (1975) observes (among other things) that they show "a slight but consistent effect" of sulfur dioxide and total suspended particles higher than air quality standards (in conjunction with a gradient for suspended sulfates).

North and Merkhofer (NAS 1975) furnish an interesting insight with respect to the relationship between different concentrations of suspended sulfates and the number of lower respiratory tract disease cases in children.* They assume that a health effect is characterized by a linear threshold relationship, and use as their model the New York Metropolitan Area with a total population of 2.13 million children 0-13 years of age. In a cleaner New York City area (with an average concentration of 16.1 $\underline{u}g/m^3$), 25% of the cases of lower respiratory disease is attributed to this pollutant. In a dirtier area (20 $\underline{u}g/m^3$), 50% of the cases is attributed to it. If each area becomes more polluted by 1 $\underline{u}g/m^3$, the percentages become 30 and 60 respectively (Figure 3).

When this percentage increase is extrapolated to the total number of cases per year at the national level wherever comparable sulfate levels obtain (in New York City this is estimated at 127 thousand per year for children 0-13 years of age), the number of excess cases is somewhat mindboggling.

Of much of the American air pollution research, NAS (B. Goldstein 1975b) finds that the studies support the

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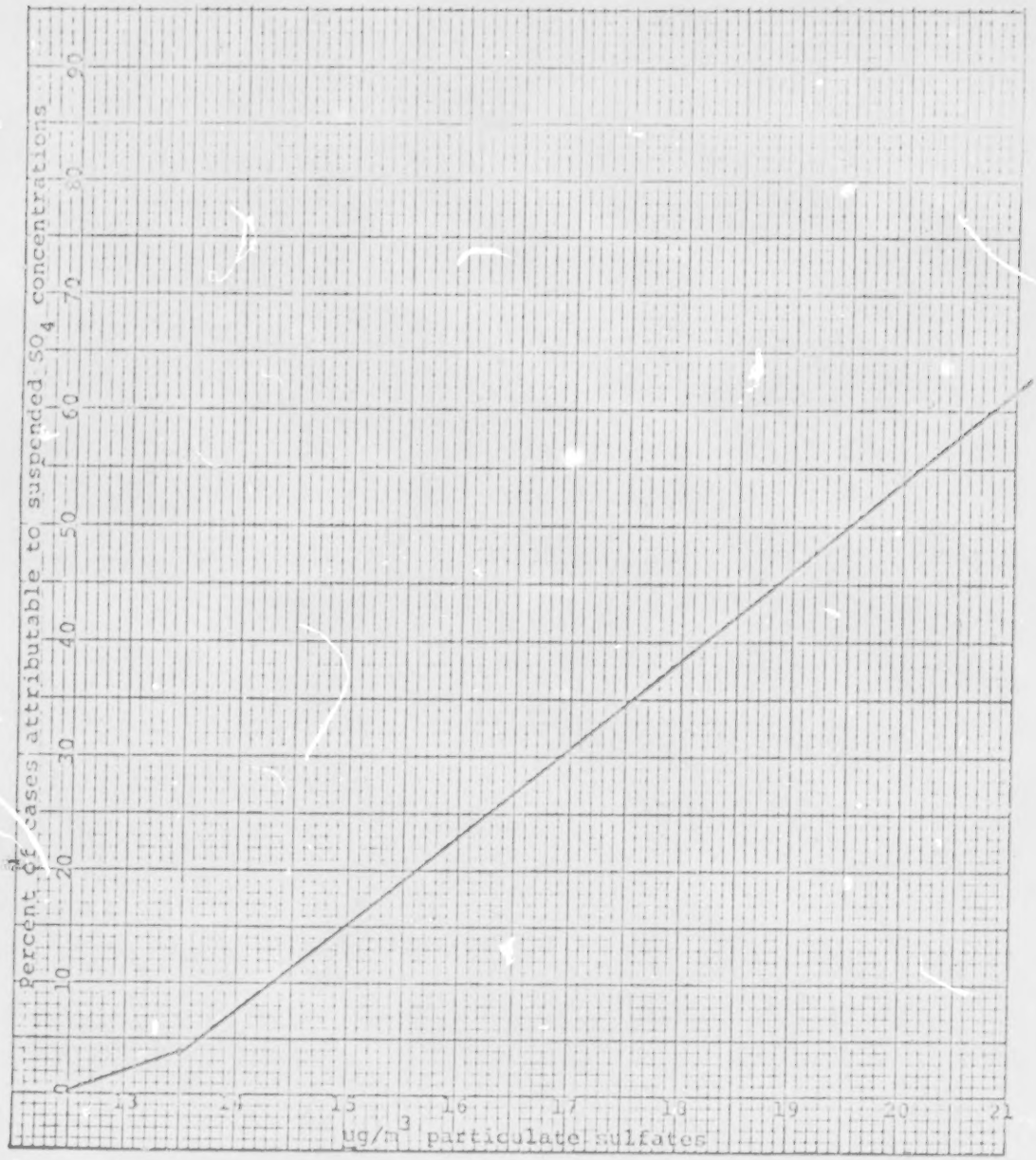
*They caution that uncertainty in the magnitude of health effects from a given increase in ambient sulfate is of the order of a factor of about 20.

Fig. 3. Lower respiratory disease & SO_4 concentration (adapted from Table 13-3, North & Merkhofer 1975)



contention that children are at risk when subjected to urban air pollution, adding that this is particularly true for lower respiratory tract infections and for asthma. He adds:

> The findings further suggest that the current air standards for sulfur dioxide and suspended particles do not have appreciable safety factors in regard to this effect on lower respiratory tract infections and for asthma. Therefore, it should be assumed that concentrations of sulfur dioxide or total suspended particles above the present standards will result in an increase in childhood lower respiratory tract infection. There is a distinct possibility that suspended sulfates are directly related to this effect.

Besides their input with respect to the health effects of sulfur dioxide and total suspended particulates at the level of the primary standards and above, it is generally acknowledged that perhaps one of the greatest contributions of the CHESS studies lies in directing strong suspicion toward the acid sulfate aerosols, for which no standards exist (Whittenberger et al. 1975).

A quite different criticism of EPA's sulfur oxide studies have to do with the fact that many were limited to middle and upper income level whites living in suburbia, and socioeconomic factors may not have been fully and consistently dealt with (Whittenbergher et al. 1975). It would seem however, that research in such areas is perhaps more meaningful, in the sense that urban and particularly inner-city areas are quite universally

recognized as being dirtier than suburban, and--if the health of suburban youngsters is impaired by pollution--urban and inner-city children are ipso facto even worse off.

But other pollutants besides the sulfur oxides and particulates--such as the nitrogen oxides--may play a role in impairing resistance to respiratory pathogens, and thus in increasing susceptibility to, and severity of, acute respiratory disease, and possibly also in increasing the risk of chronic respiratory illness.

Effects of Nitrogen Oxides

Certain Czech and Russian studies where NO_2 was perhaps the most predominant pollutant have also pointed to a relationship between such exposure and an increase in respiratory disease among children (Table 5).

EPA has reported two studies dealing with the effect of NO_2 on respiratory illness. Both of these were conducted in the Chattanooga, Tennessee, area in upper middle-class white communities (one with a relatively high NO_2 exposure*, a second with elevated suspended particulate exposure, and two controls). The first (Say et al. 1970) concerned almost 1 thousand families who in

*Aver. 0.15 (0.08) to 0.28 (0.15); 90th percentile 0.19 (0.10) to 0.94 (0.50).

TABLE 5

EFFECTS OF NO_2 ON ACUTE RESPIRATORY DISEASE
(FOREIGN STUDIES)

Study Population	Nitrogen Dioxide Concentration, mg/m³ (ppm)	Reported Effect	Reference
Czechoslovakian children, aged 7-12 years	NO_x average 0.02-0.07 mg/m³[a]	Excess of hypertrophied tonsils and lymph nodes; changes in hematologic indexes	Petr
USSR preschool and school children living near a fertilizer plant	0.32 (0.17) to 3.4 (1.8)[b]	17-fold excess of upper respiratory disease; 6- to 12-fold excess of abnormal chest films	Lindberg
USSR adolescents in vocational training at chemical and fertilizer plants	Less than 0.10 (0.053)[c]	11-27% excess of acute respiratory disease; increased blood lipoproteins and cholesterol	Giquz
Soviet children aged 8-11 years living near a ferrous metallurgic plant	Nitrogen oxides = 46.4 (87) to 93.6 (176)[d]	5-fold excess of upper respiratory disease; 3-fold excess of tonsillitis; 2.5-fold excess of atrophic rhinitis; significant lag in growth, weight, and chest circumference; decreased urinary excretion of vitamin C	Yelfimova

SOURCE: Adapted from NAS-NAE: A Report by the Coordinating Committee on Air Quality Studies, prepared for the Committee on Public Works, U.S. Senate, Vol. 2 "Health Effects of Air Pollutants," Serial No. 93-94, Sept. 1973, pp. 280-281.

[a]Other pollutant exposures includes sulfur dioxide at 0.01-0.12 mg/m³.
[b]High concentrations of sulfur dioxide and sulfuric acid were also measured.
[c]Exposure to ammonia above the U.S.S.R. maximal permissible concentration were reported.
[d]High concentrations of sulfur dioxide and hydrogen sulfide and moderate concentrations of phenol were reported.

Given the variable pollutant exposures and conditions of study, it is difficult to determine the relation of nitrogen dioxide by itself to the reported increases in respiratory disease. In each of those studies, other important air contaminants that were likely to increase respiratory susceptibility to infection (sulfur dioxide, sulfuric acid, sulfates, etc.) were also present. In broad generalities, the studies of Giquz, Shy, and Pearlman (about to be described) are consistent in suggesting an association of exposure to ambient concentrations of nitrogen dioxide of 0.11-0.58 mg/m³ (0.53-0.31 ppm) and small excesses of respiratory illnesses. These relatively small excesses can become much more important in terms of absolute numbers of illnesses when it is considered that the National Health Survey reported an average of 1.3 episodes of acute respiratory disease per person per year. A 10% increase in respiratory disease would result in 130,000 added illnesses per year in a population of 1 million persons.

1968 had children in the second grade. The objective of the study was to determine the frequency and severity of acute upper respiratory illness over a 24-week period. A relative excess in respiratory illness of 18.8% was found among families exposed to elevated NO_2 concentration, and an excess of 10.4% among families in the elevated suspended particulate exposure community. This excess occurred in all family segments including second grade children, their siblings, and parents. Areal differences could not be explained by differences in family composition, economic level, or prevalence of chronic conditons. Parental smoking habits did not appear to influence respiratory rates of the second grade children.

In the second investigation (Pearlman et al. 1971), three areas (all upper middle-class residents) with different gradients of NO_2 concentration--as above--were chosen, and infants and school children who had been exposed for 1 to 3 years were studied to determine the incidence of lower respiratory illness. Children* exposed for 2 and 3 years, and infants for 3 years to elevated levels experienced significantly** increased morbidity both in terms of the relative proportion of

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*Number not specified.

**P=.02 for 2 years; p=.01 for 3 years.

children affected and the frequency of attacks. Among infants a 10-58% excess of acute bronchitis was reported; and a 39-71% excess among 6-9-year-old children. Illness associated with croup, pneumonia and hospitalizations did not significantly differ among the three exposure areas studied.

These Chattanooga studies have received wide criticisms (primarily because of the validity of the method of measuring NO_2 concentrations). However, in NAS' view (Elliott Goldstein 1973), failing the availability of other data, decisions regarding air quality for NO_2 must rely on this work. Their significance is that exposure to atmospheric NO_2 is "virtually ubiquitous" (Shy et al. 1973) in urban U.S. life.

Effects of Oxidants

We should perhaps be reminded here of the complex atmospheric reactions between hydrocarbons and nitrogen oxides in the presence of sunlight, leading to the formation of oxidants.

As for the oxidants alone, while suggestions have been made of their effects on pulmonary function, the data so far are scant and inconsistent. As NAS (Chambers, 1973) stated:

> The epidemiologic approach has produced reasonable evidence of respiratory difficulty resulting

from life in smog-ridden areas of Los Angeles and other cities; but efforts to pin the results on a specific component or mixture of specific components have produced only a demonstration of the powerlessness of multiple correlation or multiple regression analysis in situations wherein only a fraction of the impacting chemical entities have been measured or even identified.

Of particular significance with respect to ozone is the effect of its syngergistic action with sulfur dioxide. NAS (1975) explains that sulfur oxide concentrations that are within the range of ambient concentrations not infrequently experienced in polluted areas in the U.S. may produce effects by one or more of the following mechanisms:

1. A direct effect of ambient sulfuric acid.

2. A direct effect of ambient suspended particulate sulfates.

3. An effect of sulfur dioxide potentiated by absorption onto inert particles of respirable size.

4. An effect of sulfur dioxide potentiated by metallic particles that catalyze its oxidation in the airway.

5. An effect of sulfur dioxide potentiated by simultaneous inhalation of ozone.

Inasmuch as the various sulfur oxides tend to be present together in polluted air, the effects described above may well occur simultaneously in an additive, or perhaps synergistic, manner.

Commentary. Overreaching is a charge levelled by some against EPA's CHESS program despite EPA's caveat

(in the Preface to its 1974 report) that one swallow doesn't make a summer: that one study by itself isn't conclusive, but that a series of such under disparate exposure situations showing a consistent pattern of adverse health effects does provide evidence which incriminates certain pollutants at certain levels. And now comes the realization that perhaps the CHESS studies have in fact been overcriticized (B. Goldstein 1975c). Honest differences of opinion within the scientific community there may be. But it would seem that some of this criticism--as in the past under somewhat comparable circumstances (further discussed later in this chapter)--may bear some relationship to a degree of interest or involvement in the economics and politics of emissions control. In the case of the sulfur oxides, obviously a number of energy purveyors have a considerable economic stake accompanied by powerful political clout.

It is here suggested that computing the degree of pollution control--either by underestimating the deleterious impact of emissions upon the respiratory tract and/or by placing too low a price tag on each childhood case of lower respiratory disease--may prove to be unsound and shortsighted from at least two viewpoints. The real cost of health care for a lifetime may be considerably lower with stricter controls which

embrace a sense of prevention over the long-term, which in turn leads to greater productivity over time on the part of less handicapped adults. Plus the fact that controls may perforce generate the development of desirable alternative energy policies. For both of these reasons, a greater gross national product may be the fortuitous end result.

Chronic Respiratory Disease

Despite the fact that acute respiratory infection is an important multibillion dollar public health problem and the leading cause of morbidity in the U.S., it's the chronic cumulative effects of air pollution on infants and children which should give us most reason for concern. This is in terms not just of transient respiratory infections during early life, but rather in terms of the sequelae of such conditions in later life.

British epidemiologists have perhaps paid more attention to this probelm than their colleagues here in the U.S. Several of their studies (Colley et al. 1973, Holland et al. 1969, Reid 1969, Lee 1957, and Rosenbaum 1961) have suggested a link between respiratory disease in childhood and adult life, and with the importance of airborne pollutants in contributing to such disease.

EPA completed one study (Finklea et al 1974)

designed to test several hypotheses concerning the long-term effects of pollution, including these two: (1) that residence in high pollution areas is accompanied by increases in the prevalence and/or severity of chronic respiratory disease symptoms in later life, and (2) that the prevalence of chronic bronchitis is related to the length of residence in polluted areas. This involved a group of some 45 thousand military recruits undergoing physical examination at the Chicago Induction Center during the period from December 24, 1969 to February 20, 1970. Approximately two-thirds of the recruits were less than 21 years of age; only 5% were over 24; some came from the more polluted urban core and suburban areas of Chicago; and others from rural and smaller urban areas in the outstate Illinois-Indiana area. (A third hypothesis--dealing with the combined effect of air pollution and cigarette smoking--was also tested, but is not considered here.) Prevalence rates were calculated by educational attainment, race, length of residence, and smoking habits, and mean symptom scores compiled for each exposure community.

Those who came from the dirtier area experienced "significantly higher" chronic respiratory disease prevalence rates than their counterparts in cleaner neighborhoods. Significant increases in the frequency of chronic

respiratory disease symptoms occurred with exposure lasting 12 years or more to ambient air pollution characterized by elevated annual average levels of sulfur dioxide (96 to 217 ug/m^3), suspended particulates (103-155 ug/m^3), and suspended sulfates (14 ug/m^3).

In his presidential address before the Section on Epidemiology of the Royal Society of Medicine, Reid (1969) made this observation:

> We have not, I believe, sufficiently considered the implications, as regards prevention, of the likelihood that, despite the improvement apparent in adolescence, the bronchitic child is father to the bronchitic man.

There are those who disagree with Reid's view, and though there is at present no substantial scientific evidence to support this assumption, as NAS (B. Goldstein 1975d) puts it,

> It is prudent to assume that recurrent childhood respiratory infections do play a role in the eventual development of disabling chronic respiratory disease.

Non-Allergic Airways Disease: Asthma

On the basis of the American Lung Association's 1973 statement that the prevalence of asthma among children aged 15 years or younger is estimated to be between 5 and 15%, there are in the U.S. from 2.5 to 7.5 million youngsters suffering from this chronic allergic disorder. It is said to account for almost one-quarter of all

school-day absences due to chronic conditions.

Asthma is one of the most common long-term conditions of childhood, and there's a possibility that this number may be increasing. Of course, it may merely be that more cases are being diagnosed, but--if there is genetic factor involved in asthma, and more and more asthmatic children with today's medical care are living to maturity--more asthmatic parents may be giving birth to more asthmatic offspring. Apropos of this, a Professor of Public Health at Chiba Unviersity School of Medicine in Japan reported that since 1968 the prevalence rate of bronchial asthma has been increasing among Japanese school children (Yoshida 1974). When experienced over long periods, asthma unfortunately may lead to progressive irreversible emphysema, which has been growing exponentially.

Finberg (1974), a member of AAP's Commitee on Environmental Hazards, observed that the incidence of significant asthma--either requiring the attention of a physician or those cases usually brought to attention in the emergency room of a hospital--has gone up sharply over the past twenty years. Even in the last eight years, there's been a rise in the number of patients involved and in the number of visits they make, as well as in the severity of the disturbances.

Palm (1970) concurs in this view. In reviewing asthma admissions and deaths at Children's Hospital of Pittsburgh, he noted that, for the period from 1958-1968, the number of patients hospitalized with acute asthma doubled. Fortunately, the mortality rate declined. But, as Sultz and his associates (1970) remind us, the incidence of hospitalized cases is but a fraction of the total incidence of childhood asthma and eczema.

Polluted air is particularly troublesome to allergic children because it adds just one more stressful factor with which their already sensitized bronchopolmonary system must cope. In discussing the interactions of air pollutants, defense mechanisms, and infectious agents in asthma, the Director of the Pulmonary Unit at the University of Vermont (Green 1974), pointed to the serious implications for asthmatic patients in this way:

> Clinical experience and epidemiologic data hold that attacks of asthma in children and adults are associated with acute episodes of air pollution exposure on the one hand, and with respiratory tract infection on the other.

And he explained how several insults (viral and bacterial infections and air pollution) result in a two-fold greater incidence of secondary bacterial pneumonia among allergic than non-allergic patients.

As the American Academy of Allergy's Weather and

Air Pollution Commitee (1972) states, it is difficult to separate a number of factors with respect to the precise etiology of increased or aggravated asthmatic attacks--like extremes of temperature and humidity, the more troublesome pollutants, indoor inhalants and outdoor molds, the "priming" effect exerted by ragweed, or the part geographic changes play.

Early Studies

It has been recognized for some time that following severe air pollution episodes, emergency rooms are called upon to handle more acute asthmatic patients, as are allergists in their private practices. The study by Chiarmonte and his associates (1970) at Long Island College Hospital, Brooklyn, New York, is an example of this. They found a significant* rise of obstructive bronchopolmonary symptoms related to the several days of high sulfur dioxide levels during the November 1966 air pollution episode in New York City as compared with the weeks preceding and following the episode. This increase in asthmatic attacks was more evident for the two consecutive days starting 72 hours following peak SO_2 levels.

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*Amount not specified.

Another example is the 2-year investigation of Girsh and his team (1967) with asthmatic youngsters brought to the outpatient dispensary at St. Christopher's Hospital, Philadelphia, in the late 1960's. Their object was to determine the effect of various pollutants (oxides of nitrogen, sulfur dioxide, carbon monoxide, total oxidants, and dust) and various weather factors (barometric pressure, temperature, relative humidity, wind speed, wind direction, and precipitation) on patients seeking help--particularly on each "noteworthy" day. A day was considered noteworthy when the concentration of pollution reached above the highest tenth percentile of scores observed during the past year's study period. Days when masses of high barometric pressure moved over the City of Philadelphia were also specially charted; so, too, were stagnant air, low wind velocity, and poor visibility.

Herewith a summary of Girsh's findings:

(1) A three-fold greater incidence of asthma occurred during days of high air pollution;

(2) a four-fold greater incidence occurred during days of high barometric pressure;

(3) stagnant air and high air pollution increased the rate nine times;

(4) the peak incidence of asthma occurred just after ragweed season in October and November, with May the third month in order.

Further, Girsh reported that the occurrence of

stable weather conditions with stagnant air over Philadelphia seemed to correlate with peak incidences of asthma. There are indications, he believes, that the increased air pollution associated with these weather conditions is a causative factor. As a possible explanation for the increased frequency of asthma during days of significant high barometric pressure, he conjectures that there may be increased concentrations of pollutants not at present being recognized and measured; and, as another possibility, that high barometric pressure alone may be the triggering factor.

One of the early studies of the effect of moderate air pollution on childhood asthma in this country was conducted in Erie County, New York, by Sultz and his associates (1970) in the 15-year period from 1956-1961. They studied four areas where total suspended particulates ranged from a low of less than 80 to a high of over 135 $\underline{u}g/m^3$, with two intermediate ranges of from 80 to 100 and from 100 to 135 $\underline{u}g/m^3$. The total child population under 15 years of age in Erie County (over 1/4 million) was divided into four social classes, and the incidence of cases requiring hospitalization during the course of illnesses (eczema cases were studied, too) was determined.

Sultz reported that the incidence rates of

hospitalized cases with each condition produced a definite gradient by air pollution level.* The incidence rates for hospitalized cases of asthma by social class and air pollution level revealed a generally consistent increase by air pollution level within each of the four social classes. On the other hand, there was an irregular pattern of variation in the incidence of hospitalized asthma patients by social class within air pollution levels.

On this latter point, it has been observed that residence is a function of social class, i.e., 78% of the poor children in Buffalo lived in areas with particulate averages of 100 ug/m^3 or more, while 90% of the more affluent children's homes were located in an area where particulates averaged less than 100 ug/m^3.**

Sultz' findings reaffirmed two other widely held medical opinions: (1) that the younger the child, the greater the risk of asthma (more than 3 times as many

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*An average annual incidence rate of 32.4 hospitalized asthma cases per 100 thousand population at risk was found at the lowest air pollution level in comparison to a rate of 50.6 at the highest.

**It would seem appropriate to point here to the fact that, in EPA's population at risk based on 1974 aerometric data (as discussed in Chapter II), almost 7.5 million children--not necessarily asthmatics--from 0 to 19 years of age are subject to total suspended particulate averages above 91 ug/m^3. If 10% were asthmatics, this would make 750 thousand at risk.

children under 5 as between 5 and 15 are hospitalized); and (2) boys are more susceptible than girls.

More Recent Studies

A more recent report deals with the relationship of asthma emergency room visits to air pollution and to weather in New York City. I. Goldstein and Block (1974) evaluated data from hospitals in Harlem and the Bedford-Stuyvesant area in Brooklyn for the period September through Decmeber 1970. With temperature held constant, there was a positive correlation between asthma visits by both children and adults and SO_2 in Brooklyn but not in Harlem (the difference appears inexplicable, except for the pattern of industry-polluted air from New Jersey passing over Manhattan first). They repeated the analysis for children under 13 in September through December 1971 at Brooklyn's Kings County Hospital, and replicated the 1970 findings. On the 12 high pollution days with sulfur dioxide concentrations at 0.108 ppm (standard=0.03 ppm), the increase in emergency room visits amounted to 50 to 90%.

In EPA's Rocky Mountain study (Finklea et al 1974), it was contended that asthmatic children in dirty areas had "significantly"* more total acute lower respiratory illness, croup, and bronchitis than those in cleaner

*Amount not specified.

communities in terms of sulfur dioxide and suspended sulfate pollution. Indeed, they found "strongly suggestive evidence" that excess lower respiratory illness in asthmatics can be linked to annual average sulfur dioxide levels as low as 67 $\underline{u}g/m^3$ accompanied by elevations of annual average levels of total suspended particulates (99 to 115 $\underline{u}g/m^3$) and intermediate suspended sulfates (6 to 7 $\underline{u}g/m^3$).

Other CHESS diary studies with asthma panelists (some of whom were under 16 years of age), have been made in the Salt Lake Basin area and in New York City (Finklea 1974). Among asthmatics in the west, the daily attack rates were more consistently correlated with colder outdoor temperatures than with any measured pollutant. When an analysis was made of the daily pollution concentrations within two temperature ranges--30 to 50°--for sulfur dioxide, total suspended particualtes, and suspended sulfates, asthma attack rates were consistently found to be most closely related to stepwise increases in the levels of suspended sulfates. There was virtually no relationship between sulfur dioxide and the attack rates. Also the effect of cold temperatures was considerably stronger than that of the sulfates, and the pollution threshold for the asthma response was much higher in colder than in more moderate temperatures.

The difference between western and eastern asthmatics--as far as attack rates is concerned--is one of weather: attack rates in New York City were generally higher on days with more moderate temperature.

Though NAS (1975) has certain reservations concerning EPA's Salt Lake and New York City asthma findings, they summarize that "reasonable preliminary conclusions" are that they do provide support for the association of air pollution with asthma attacks, and that they further implicate suspended sulfates as an "important deleterious component" of polluted air.

Contrary to the positive correlations mentioned above between various pollutants and asthma, Schoettlin and Landau (1961)--in Los Angeles--found the association between increased atmospheric sulfates and increased asthmatic morbidity much less impressive for children than for adults. There are other negative reports such as those of Lewis, Gilkerson, and McCalden (1962), who observed no association between the daily levels of air pollution in New Orleans and the frequency of visits to Charity Hospital for the treatment of asthmatic episodes. Also the Rao group (1973) experience in Brooklyn, New York, with respect to both sulfur dioxide and smoke shade, and that of Ribon and his team (1972), who report that only temperature change was a factor in the number of visits

to emergency rooms or admission to hospitals, and not pollutants such as carbon monoxide, sulfur dioxide, oxidants, and smoke shade.

EPA faults much of the work in connection with air pollution and asthma on one or more of the following grounds: (1) defective monitoring; (2) inaccurate or insensitive procedures for quantifying morbidity; (3) inability to reasonably estimate dose-response relationships between air pollution and health effects; and (4) inability to separate effects of air pollutants from weather parameters.

However, they believe that two of their more recent studies have succeeded in ruling out all factors except pollutants in asthma attack rates. The first such study, with a team headed by Cohen (1972), involved a total panel of 20, of whom there were just 4 under 16 years of age. They reported daily symptoms for over 7 months while air pollution and weather parameters were monitored. This panel was intermittently exposed to high levels of pollution because their homes were located within 1/2 mile of a coal-fueled power plant which emitted substantial quantities of particulates, sulfur dioxide, and oxides of nitrogen. No single pollutant could be singled out as the prime cause of asthma episodes, though suspended sulfates showed the strongest association with

attack rate after the effects of temperature were removed. Cohen comments that this finding may reflect only the unusual positive correlation of suspended sulfates and temperature, or may reflect the irritative properties of sulfuric acid mist. Significant correlations were found between reported attack rate and temperature and between attack rate and pollution levels after the effects of temperature had been removed from the analysis.

These temperature-independent air pollution effects, Cohen observed, occurred at levels of pollution commonly found in large cities--such as New York and Chicago--and appeared greater at moderate than at low temperatures.

NAS (1975) finds fault with this study on several grounds (primarily because of the small number of cases studied), but believes that the general findings "appear" to support the contention that power-plant effluents are instrumental in potentiating asthma attacks.

Rall (1974) also faults the Cohen study. His view is that temperature had more effect than air pollution, and that all pollutants were associated with asthmatic attacks.

Quite the opposite view is expressed by Finberg (1974). He believes that Cohen's study strongly implicates sulfate particulate matter as "the most significant

correlate of clinical disease."

The second recent EPA study, by Hammer et al (1974), "clearly" relates acute episodes of croup among asthmatic children in the southeastern part of the U.S. with increased particulate air pollution exposure: This is the statement of AAP's Chairman of their Committee on Environmental Hazards (Wehrle 1974).

Besides the irritant effects of SO_2, TSP and sulfates, a number of investigators have commented on the ability of the insoluble gases--such as ozone and NO_2--to penetrate along with the respirable gases (such as SO_2) to the respiratory bronchioles and alveoli (Knelson et al 1974). High oxidant levels also have been reported to affect the frequency of asthmatic attacks (AMA 1973).

In summary--as Finberg (1974) expresses the situation quite succintly--

> The relationship may not be a simple one of cause and effect; but, clearly, whatever else is going on, air pollution aggravates asthma and probably almost all respiratory disease.

Mental Illness and Behavior Change

The association between air pollution and physical health is perhaps more readily apparent than the impact of dirty air on mental health. But, just as physical health is a continuum from predisease to terminal conditions, so, too, is there a wide spectrum of difference

between a state of mental wellbeing and frank psychosis. The indirect and delayed sequelae of traumatic environmental situations may have an unfortunate effect on children's adaptive responses, unfortunately handicapping them at the beginning of their lives (Dubos 1972). Many such effects are ill-defined, but there are airborne pollutants which have a very direct effect on the central nervous system (CNS).

The three elements in air which appear to affect children's mental health most profoundly are carbon monoxide, mercury, and lead. These potent neurotoxic agents can produce serious and irreversible damage and behavioral change.

There's a "growing tendency"--the National Institute of Mental Health (NIMH 1972) points out--to be concerned with low levels of carbon monoxide, and particularly with the psychological consequences of exposure to everyday levels. This applies especially, but is not restricted to, young infants and fetuses, and thus to pregnant women. Children riding in cars or playing near heavily travelled highways can suffer a degree of hypoxia (oxygen lack) and consequent brain damage caused by carbon monoxide. They can also be affected by the carbon monoxide in cigarettes and by faulty heating systems, as covered in Chapter III dealing with "Indoor Pollutants."

While the much-publicized cases of mass poisoning

from mercury have occurred from eating fish contaminated by effluents discharged into water or by fungicides sprayed on grains and eaten by fowl, cattle or hogs, and then by humans, airborne mercury is a potential threat: it is continually recycled from air to water and soil, and back again to the air.

Lead Poisoning

The airborne element which is perhaps most dangerous to the mental health of children is lead. The principal source of airborne lead is, of course, auto exhausts. Lead from car exhausts contaminates water supplies as well as air, and affects crops at distances as far as 50 meters from highways, depending on weather conditons and traffic volume (WHO 1972b). Thus lead may be drunk as well as inhaled.

Lead reaches the pediatric population through a number of pathways: it crosses the placental barrier, and may affect the embryo and fetus; it is ingested by toddlers who develop pica (a craving for oral satisfaction from unwholesome objects) and satisfy their craving both indoors and outdoors; it is also ingested by pica-free youngsters as they play in the dirt and engage in a great deal of hand-to-mouth activity.

Lead poisoning has been likened to the scourges of typhoid or cholera. Indeed, it has been said that

lead poisoning "actually represents a greater hazard to the brains of urban children, if not to their life expectancy, than did polio or measles at their worst" (Finberg, 1970).

Lead poisoning early in life may doom a child to lifelong illness. In 25 to 40% of the cases of acute poisoning, CNS damage is likely to occur; in repeated episodes of poisoning, the chance of serious and irreversible damage is virtually 100% (Perlstein and Attala 1966; Chisolm and Harrison 1956). The brain can be so seriously crippled that only a vegetable-like existence is possible, each such institutionalized child costing society about 1/4 million dollars for his lifetime care, according to the National Academy of Sciences (NAS, 1972). If brain injury isn't this extensive, some degree of mental retardation and learning disability frequently ensues, with behavioral difficulties and emotional instabilities also--presenting long-term burdens not only for parents but for doctors, teachers, and the public purse. (NAS "roughly" estimates that special schooling for each case involving moderate permanent brain damage amounts to $18,000.)

Besides CNS pathology, there may be impairment of other organs and systems, such as the hematopoietic, hepatic, renal, and skeletal, with blindness a possible aftermath.

Low Level Lead Poisoning

One of the difficulties in coping with the management of lead poisoning--the "silent epidemic" (Rothschild 1970) because of the non-specific nature of the symptoms which so often mimic many other disease symptoms--has been the identification of the condition early enough to check the development of serious, irreversible pathology.

Because of the numbers of children known or suspected to be affected by lead poisoning--and because the condition is preventable--the Surgeon General of the U.S. Public Health Services (PHS) in 1970 issued a policy statement, calling for the screening of all children from 1 to 6 years of age living in old, poorly maintained homes (in the so-called lead belt areas). The prime goal of screening programs, the Surgeon General stated, is prevention of plumbism (lead poisoning), with such prevention being achieved through early detection of youngsters with what he referred to as "undue" absorption of lead, followed by remedial action before the state of overt poisoning is reached.

The Surgeon General's recommendation was that a blood lead concentration of 40 ug/100 ml. of whole blood, as validated by the dithizone technique, and determined on two separate occasions, be considered evidence

suggestive of undue absorption of lead--either past or present.

In the intervening five years since the Surgeon General's policy statement--as a result of nationwide surveys and screening programs, and as a result also of a number of animal and clinical studies concerning the effects of low lead levels--new insights have been gained about the lead poisoning problem. These concern the following:

(1) desirability of establishing a Maximum Daily Permissible Intake (MDPI) for young children;

(2) wide prevalence of chronic low level poisoning;

(3) reduction of "undue" absorption level;

(4) significance of airborne lead in dirt and dust;

(5) neuropsychologic manifestations of low levels.

Desirability of Establishing MDPI

When, in 1970, the Surgeon General issued his policy statement, lead paint--and particularly the ingestion of lead paint chips--was considered largely responsible for pediatric lead poisoning. Indeed, the serious public health problem of overt lead intoxication from eating lead-based paint tended to obscure low level lead toxicity which may be related to atmospheric lead (Knelson 1974a).

Because of its toxicity and its particular affinity for children, and because lead is such a ubiquitous metal found in air, food, and water, as well as in many man-made artifacts (certain pewter and pottery articles, newspaper print, certain toys, crayons, certain canned foods, etc.), it was felt desirable to establish an amount which could be considered a tolerable dose from all sources for small children. In 1971, an HEW Committee established this at 300 ug for youngsters 1 to 3 years of age (King). Others have estimated the level for preschoolers to be between 70 and 500 ug (Lepow et al. 1974).

Wide Prevalence of Low Level Lead Poisoning

In the late 1960's and in 1970-1971, prevalence of undue or increased absorption of lead was found among an alarming proportion--something on the order of 25% or more--of children in lead-belt areas in seven metropolitan centers. This finding resulted in assessing the magnitude of the condition among children living in old neighborhoods as matched by few, if any other, pediatric public health problems (Lin-Fu 1972).

Hard upon this insight several others emerged: that undue absorption of lead is not confined soley to the children residing in dilapidated structures in inner city. Indeed, as Appendix B-1 reveals in the more than

a dozen publications listed, increased absorption is a somewhat universal American condition. It exists in virtually all regions of the country, and under various circumstances, as follows:

in "outer" urban neighborhoods,

in medium and upper income families,

in homes in good condition,

in small, medium, and larger communities, and

in suburbs and rural areas.

Evidence on these points is derived from such examples as these (delineated further in Appendix B-1).

Screening in Baltimore in 1966-1970 revealed that 43.3% of children with levels of $\geq$60 ug/100 ml. came from outside inner city (Lin-Fu 1973). A similar situation obtained in Philadelphia where approximately 50% of the reported cases of childhood lead poisoning were located outside of inner city; indeed, some lived in census tracts where property values were equal to or greater than average property values of most dwelling units in the City of Philadelphia.

In a study by Needleman and Shapiro (1974), 43% of young white preschool students living in old houses, but in good repair, had high dentine lead concentrations in their deciduous teeth (indicating bodily storage of lead).

Again, Fine et al. (1972), in examining lead absorption in children 1 to 16 years of age in 14 Illinois cities of intermediate size--10 to 150,000 population--found that 18.6% had blood lead levels >40 ug/100 ml.

In comparing a rural community in western Connecticut and adjacent New York State with urban Hartford--it was Cohen's (1973) experience that 9% of rural and 23% urban children showed blood lead levels of ≥40 ug/100 ml.

True, the same prevalence rate found in inner city may not apply in all regions of the country. (However, it should be mentioned that the 25% figure which Lin-Fu reported as existing in 7 metropolitan centers was arrived at in the main on the basis of a single--and not a confirmed--blood test; what fraction of these were later confirmed is unknown; it could have been less.)

In nationwide surveys of most recent date (1973-1975), conducted by HEW and involving over a million children, the rate for confirmed tests is considerably lower, approximately 6% (Lin-Fu 1975); however, to the extent to which the "substantial" number (which she mentions was 24% in FY 1973 and 14% in FY 1974) with initial higher readings who failed to show for a second test would have been positive, to that extent the 6% figure should be increased.

On the basis of housing conditions alone (meaning

lead paint exposed surfaces producing the deposition of flakes frequently ingested by youngsters), the U.S. National Bureau of Standards estimated that 600 thousand children aged 1 to 6 years have increased lead absorption (Gilsinn 1972). With a total population of 20 million youngsters under 5 years of age in the U.S., and a prevalence rate of 6% with blood levels ≥40 ug/100 ml (based on HEW's sample of 1 million youngsters referred to by Lin-Fu), it may be conjectured that somewhere in the neighborhood of 1 million youngsters are exposed to possible undesirable lead absorption from a number of sources, not lead paint alone.

Reduction of "Undue" Absorption Level

The Communicable Disease Center (CDC) of PHS now carries on where Steinfeld left off when the post of Surgeon General was abolished. In its March 1975 statement, CDC lowered the blood lead level which they consider reflects a state of increased absorption. Along with blood lead, they also recommend a determination of the erythrocyte protoporphyrin--EP--level not only because it is a simpler laboratory procedure, but because it is a better index of potential toxicity, and is usually elevated before clinical symptoms begin. They consider that there are three grades of undue absorption: minimally elevated, moderately elevated, and extremely

elevated, as follows:

Test	Minimally Elevated	Moderately Elevated	Extremely Elevated
Pb	30-49	50-79	$\geq$ 80
EP	60-109	110-189	$\geq$190

This updated definition of risk from undue or increased absorption by CDC thus represents a 25% reduction from the 40 ug/100 ml. recommended by Steinfeld five years before. It is consistent with the findings and recommendations of a number of researchers who have recently reported on their studies dealing with CNS effects of low lead levels--notably David (1972, 1974a), Millar et al. (1970), Perino and Ernhart (1974), Roels et al. (1975), shown in Appendix B-3. All of these investigators stress the desirability of a reduction in the level, so as to provide a greater margin of safety. Indeed, both David and Roels et al. advocate a further 15 to 25% reduction from the 40 ug; they believe that 25 ug should be the cutoff point.

CDC's recommendation is somewhat consistent also with EPA's position statement of 1972, in which they recommended a level of 30 ug for pregnant women--of significance because lead crosses the placental barrier, as already stated--and for newborns.

It is noteworthy that even the youngsters designated as "minimally elevated" by CDC--those with blood levels of from 30-49 ug--are considered at risk: CDC recommends that these minimally elevated children be carefully and regularly checked until they reach their sixth birthday. Thus, the catchment group for undue or increased absorption--instead of 1 million children with blood levels ≥40 ug--increases with the inclusion of that proportion of youngsters who are minimally elevated beginning with a blood level of 30 ug. What this increase might amount to is unknown.

Airborne Lead in Dust and Dirt

Based on her determination of the mean lead level of Hartford street dirt at 1200 ug/g, and of housedust at 11,000 ug/g--and on the assumption that a youngster at play may be putting his fingers or hands in his mouth 10 times per day (each time ingesting about 10 mg. dirt)--Lepow et al. (1974) concluded that it is quite possible for a young child during play in a heavily leaded environment to ingest excessive quantities and accumulate an increased body burden. While these levels of street dirt lead are consistent with the findings of street dirt in 77 midwestern cities (Hunt et al. 1971), and of another investigator with housedust (Sayre et al. 1974), actually

Hartford street dirt lead is lower than that in many American communities: NAS (1972) states that concentrations often exceed 2000 ug/g.

Even a moistened lollipop dropped to the ground can pick up 0.5 g of dirt, which--if the dirt contained 0.5% lead (highly possible if not probable in many parts of the U.S.)--would yield 2500 ug of lead, an amount greater than that routinely found in the daily diet of children (EPA 1973).

It has been estimated that, if a 1-year-old scoops up daily into his mouth only 1/4 to 1/2 teaspoon of residential dustfall, he would within about 8 months suffer from lead poisoning, reaching a blood lead level of 60 ug. If a child with pica consumes the same amount near a roadway, he would be expected after one month to reach a dangerously high blood lead level, 80 ug (EPA 1972).

Evidence from several other studies besides that in Hartford reveals that high rates of excessive lead absorption have occurred in young children regardless of exposure to paint. For example, in Newark, N. J. (EPA 1973), high traffic density significantly affected the degree of absorption among children also exposed to paint. Children living within 100 ft. of major traffic arterials exhibited twice the frequency of undue lead absorption as those whose residences were located at a

distance of 100 to 200 ft. or more from the roadway. And, in this latter group, the extent of absorption increased with an increase in weekday traffic densities.

In Philadelphia, when dentine lead levels were used as an index of lead absorption, striking differences were observed between children with greater exposure to airborne lead (both from an industrial source and from automotive emissions) and those living in a community free of industrial lead sources (Shapiro 1972 and Needleman and Shapiro 1972, 1974).

In New York City, the Bureau of Lead Poisoning Control reported in 1972 (EPA 1973) that only 50% of children with blood levels between 25 and 44 ug/100 g could be associated with living in homes containing pealing paint of 1% lead; nearly 20% lived in homes in which no pealing paint was identified.

A similar situation was found in Multomah County, Ore., where 50% of the children with elevated blood lead levels lived in housing where lead paint was not considered accessible to them (EPA 1973).

Again, in El Paso, Texas, among a group of children living near a smelter and where lead paint was not a significant factor, 90% of the 1- to 5-year-olds had levels 40 ug. Of special significance is EPA's statement that soil lead levels in the vicinity of the smelter

are not materially different from levels of lead in dirt and soils found in many urban streets and parks (EPA 1972).

NAS (1972) concurs in the view that the high concentration of lead in urban air and on the surfaces of parks and streets constitutes a source of intake additional to the usual dietary sources, and may be a significant source of lead intake for the 25% of children in inner city areas who have dangerous blood lead levels despite no detectable lead paint in their homes. They state that assuming the validity of the relation between daily lead intake and blood lead content, the daily ingestion by a child weighing 10 kg of 0.41 g of street dust with a lead content of 2000 ug/g would result ultimately in a blood lead concentration compatible with clinical lead poisoning, even without allowing for additioanl lead acquired by inhalation, from normal dietary sources or from coincident ingestion of leaded paint.

Thus from the above--as well as from numerous comments in the literature--it would appear that professional opinion has in the main swung away from the belief that paint and paint alone represents the sole source of lead exposure for young children, and that airborne lead both indoor and out plays an important contributor role (more

on lead indoors in Chapter III). WHO (1972a) reminds us that in addition to being a source of immediate inhalation exposure, airborne lead is important for the transport of the metal to other areas and media.

Indeed, in some cases, airborne lead may prove the factor which steps up the elevation to a highly dangerous level and places the child in serious jeopardy. An important consideration may be that the small particle size and greater surface area of dust as compared to paint chips may enhance its absorption (Lin-Fu 1975); also that the small particle size easily reaches the alveoli (Habib 1970).

Neuropsychologic Manifestations of Low Lead Levels

Two tables (see Appendix B) contain rather fulsome details concerning recent studies which have appeared in American and British literature investigating the impact of low lead levels on the young. One of these (B-2) comprises published reports of experimental work with animals; another (B-3) comprises reports of studies in children.

Animal Studies. Appendix B-2 dealing with animal studies furnishes the highlights of ten of these investigations. Whether the researchers chose mice, rats, sheep, or monkeys for their experiments, the reports show complete

consistency in the finding that young--in contrast to mature--animals are uniquely susceptible to low lead level intoxication, and manifest CNS dysfunctions comparable in many respects to those experienced by their human counterparts. And one study (Carson's) points to the deleterious effects of maternal exposure on the fetus.

In the rhesus monkey studies by Allen et al. (1974), for example, exposure to lead acetate early produced hyperactivity and insomnia in infant but not in adolescent and adult monkeys, even though high blood lead levels and histopathological changes in liver and kidneys were found in the latter.

In lambs, Carson's team (1974)--which exposed pregnant sheep to only moderately increased blood lead levels--demonstrated slow learning of nonspatial, visual discrimination problems when the young reached 10 to 15 months of age. None of the pregnant ewes exhibited clinical evidence of toxicity during or after the exposure period.

In rats exposed to lead since birth--first in mother's milk and later in their solid diet--a study by Michaelson et al. (1974) reported hyperactivity, aggressiveness, tremors and stereotyped behavior. Also found: a significant decrease in brain dopamine in the lead-exposed animals, suggesting a biochemical basis

for the CNS dysfunction.

In mice, similarly, Silbergeld's group (1974) observed developmental delays and hyperactivity in sucklings exposed to lead from birth in mother's milk and water. They domonstrated, too, that--like hyperactive children--the affected mice responded to treatment with amphetamine and phenobarbital.

Human studies. Appendix B-3 dealing with studies on the effects of low level lead in children contains the relevant details of work reported in the last several years by 15 researchers investigating essentially the effects on the CNS. It will be noted that the study populations differ in a number of respects: geographical location, age, socioeconomic status, neuropsychologic state, length of exposure, etc.; so do the methodologies employed--obviously some being more capably designed and more meticulously controlled than others. Likewise the findings, dealing with a number of manifestations of neurologic and psychologic defects, vary in both the degree and type of impairment apparently caused by or associated with low level lead poisoning.

A brief summary of the results reported is virtually impossible, except for the very broad nonspecific observation that--of the 15 investigators--12, on the basis of their findings, consider that a low level of lead is

definitely deleterious to young organisms in impairing intellectual performance and neurological and motor function. Three others hold the opposite view, and one group--though unable to identify pathology from airborne lead alone--opts for chelation therapy in asymptomatic cases to avoid possible danger.

Four of the investigators (Albert et al. 1974); Kotok 1972; Millar et al. 1970; and Perino and Ernhart 1974) focussed their research primarily on the effects of lead on mental functioning, IQ performance, and general cognitive, verbal, and perceptual abilities. Five (Baloh 1975; de la Burdé and Choate 1972 and 1975; David 1972 and 1974; Feldman 1973; Roberts et al. 1974) dealt in the main with an examination of gross and fine motor behavior, and hyperactivity. Three checked both mental functioning and behavior (Landrigan et al. 1975; Lansdown et al. 1974; and McNeil and Ptasnik 1975). And three dealt with other aspects (Beattie et al. 1975; Pueschel et al. 1972; and Roels 1975).

Not only is a more specific brief summary difficult, but a comparative evaluation of the validity and reliability of the findings is fraught with virtually insoluble problems. Criticisms have been levelled at some of the studies on a variety of issues, involving number of subjects and controls, suitability and adequacy

of tests both for lead levels and for determination of the degree and kind of impairment, and other methodological defects. Criticisms have also been made of the soundness of the interpretation of results. All these published criticisms which the literature search has revealed are included in Appendix B-3.

Commentary

In the past year and a half, controversy seems to have peaked--particularly in viewpoints expressed in a number of issues of Lancet--concerning many of these studies, notably those of de la Burde and David, and most especially those of Lansdown and his group and the Landrigan team. Battle lines would appear to be fairly clearly drawn between believers and non-believers in the deleterious sequelae from a chronic state of low level lead absorption. The issue may not be primarly scientific; it would appear to be more purely political, in the sense that economic factors are involved, with heavy infiltration by industrial interests--if the view expressed in a letter (Needleman et al. 1975) signed by eight men (six researchers and two industry workers) has validity. Here is the gist of their evaluation of the situation.

> We have noted that reports and commentary on lead toxicity tend to sharply divide between those who are alarmed that 'low level lead' exposure is accompanied by adverse health effects, particularly in neuropsychological performance, and those who

> argue that no case has been made for low-level lead exposure as a hazard.
>
> We could find no statement, report, or study by an industry-sponsored scientist, whether in-house grantee, or academic consultant, which frankly acknowledges that low-level exposure is hazardous. Those reports of toxicity due to low-level lead exposure, on the other hand, tend to come from departments of public health, pediatrics, or environmental science. This observation appears well supported by the recent literature and by views expressed at three major meetings [one national and two international--dealing with health effects of lead and other environmental pollutants].

Others besides Needleman and his colleagues have been exercised over industry's involvement in the assessment of the contribution of atmospheric lead to the total body burden. This controversy has been smoldering for a number of years, and most particularly since the publication in 1972 of Airborne Lead in Perspective, representing NAS' analysis made at the request of EPA. Even before this publication appeared (as a matter of fact in 1970), the compositon of the Panel on Lead of the Committee on Biological Effects of Atmospheric Pollutants responsible for the document was criticized for being "top heavy" with industry scientists (Gillette 1971). The final report proved bitterly disappointing to many non-industry-associated professionals, and has been heavily attacked (Boffey 1975). Though it cannot be alleged that this NAS appraisal was solely responsible for slowing up any federal regulations of lead emission controls, EPA needed

this body--as the "supreme court" of science--to support its contention concerning the adverse health effects of airborne lead, as expressed in its 1972 Position Paper. Such NAS support EPA failed to get.

This clash between viewpoints is all too reminiscent of the now historical--and often bitter and exceedingly protracted--conflict between the tobacco industry and health-concerned professionals concerning the harmful effects from tobacco smoking. It took many years and many studies by many investigators to win over the powerful protests of the commercially motivated industrial scientists. Most unfortunately, however, in the case of lead (except for those adults occupationally exposed), the population at greatest risk is small children, small children who in their "work places" at play near the ground (or floor) are unwittingly exposed to a greater concentration and absorption of lead through inhalation and ingestion. It is earnestly to be hoped that the pro bono publico adherents will be able to muster adequate strength in a shorter period of time to protect youngsters' mental health and learning abilities and performance over a lifetime.

Growth and Development

Research in this country concerned with the

pediatric aspects of air pollution has in the main dealt either with its effects on the respiratory tract or on the central nervous system.

With the exception of studies dealing with lung function and development, and one with athletic performance (Wayne et al. 1967), virtually no attention has been paid by Americans to any impact airborne pollutants may have on children's general growth and development. Indeed, literature research to date has revealed only no American studies on the subject, but just three allusions to this aspect, all referring to work outside the U.S. (AAP 1970, NAS 1974, M. Cohen 1974).

Appendix C lists the highlights of some 17 studies concerning the effect of air pollution from various sources on children's growth and development conducted in certain European countries (published in Czech, German, and Russian periodicals from the years 1966-1973) and 4 published in Japanese journals for the period 1968-1973. In general, besides evaluations of lung function, the assessment of growth and development by these overseas researchers is based on one or more of the following criteria: (1) blood determinations (hemoglobin and red cell values, protein saturation, etc.); (2) Xrays of wrists and hands; (3) body height and weight measurements; and (4) physical strength and

endurance as determined by athletic performance of various types.

Limitations Inherent in Abstracts and Translations

In the main, English abstracts were all that was available of most of the studies described in the Growth and Development table; translations of complete reports were obtainable for just five of the studies. (One exception is a citation from a book author's description of a study dealing with pollution from lead.)

Often the abstracts--consisting as they do only of relatively brief summaries--contain inadequate information concerning many important aspects--such as number and age of test subjects, type of pollution and concentration of various elements, tests employed, etc. In a number of cases, such inadequate information makesit difficult to form a competent judgement as to the validity of the reported findings. The semantics involved is another complicating factor. So, too, is the fact that some of the diagnostic tests used are unknown in the U.S. Perhaps the most significant limitation is in the failure to separate out--or to include mention of--socioeconomic factors which may have played a part in contributing to the deleterious effects reported.

Difficulties of Extrapolating to American Children

Besides the limitations cited above, there are other difficulties involved in attempts at extrapolating from the European and Japanese studies. These have to do essentially with two factors: ethnic and/or racial differences with response to pollution--this may apply in particular to Oriental children (NAS 1975)--and methods and devices employed in gathering aerometric data and in documenting pollution concentrations over time. These two differences plus the problem of determining the variables which may have played a part in the findings, as well as the national and cultural differences between countries, make extrapolation to the American scene difficult. Nevertheless, and with these caveats in mind, it would seem prudent to examine this material with a view to determining the significant implications, if any.

Highlights of Reported Findings

From the data available, four of the European studies--those of Grosser, Pelech, Petr and Schmidt, and Symon and Petr--would seem to stand out as superior. Grosser (1970) apparently gives very serious consideration to a number of measures of socioeconomic standards and to a determination of some thirty different characteristics for assessing changes as a result of the effects of

airborne pollutants. Pelech (1970) also separates out the influence of different social environments, as do Petr (1966-1967) and Symon (1970).

In sum, these investigators believe that pollution alone (from SO_2, NO_2, dust, and arsenic) is responsible for an adverse effect on children's growth and development as manifested by retarded bone maturity, certain hematological characteristics--such as a reduction in red cells and hemoglobin--and an excess of hypertrophied tonsils and lymph nodes. The remaining investigators all view air pollution as inimical to children's growth and development, including one study where lead was the pollutant and scholastic ability seemed to be affected as well. Pollutants reported by the Russian investigators include those mentioned above plus hydrogen sulfide, ammonia, and phenols.

To assess the effect of pollution on growth and development, two of the Japanese researchers utilized Xrays to measure osseous development; two checked athletic performance (mostly certain running and jumping tests), and other measures. Their findings are disparate: two reported negative or mixed results; two others reported that pollution significantly affects capacity for endurance and athletic abilities. Besides SO_2 and dust, pollutants mentioned by the Japanese include fluorides.

Akin to some of the Japanese findings with respect

to the effect of air pollution on athletic ability is the work in this country above referred to, reported by Wayne and his associates. This dealt with the effect of photchemical oxidants on a group of healthy high school country runners in Los Angeles. The records of over 100 boys were checked to determine the per cent of those who failed to improve when their running time was compared to that at the previous meet on the same course (on the basis that running time tends to improve throughout the season).

The highest correlation to team performance was found to be the oxidant level in the hour before the race, with CO, temperature, and humidity showing no relationship to performance. Wayne summarized as follows:

> The specificity of the effect to a biologically meaningful time and the very high correlation of oxidants are convincing evidence of a cause and effect relationship. The mechanism by which oxidants affect performance may be directly physiological or may be decreased motivation due to discomfort.

Significance of Overseas Reports

AAP's Committee on Environmental Hazards over five years ago (1970) commented on the "relative lack of serious consideration of the [air pollution] problem in this country." In the interest of prudence, should this lack of serious consideration be allowed to continue?

Pollution exposure in some of the areas in Europe where air pollution research has been conducted may be more serious than in most parts of the U.S., but in other cases this may not be the case. For example, NAS (1974) refers to the very low nitrogen oxide concentrations at which Czechoslovakian investigators reported hematologic changes, and also calls attention to another instance in which retarded physical development was reported to have occurred in areas where measured exposures to SO_2 and nitrogen oxides were below U.S. and U.S.S.R. standards.

Again, many of the Japanese studies have tended to yield evidence of an effect of much lower concentrations of pollutants than have American or European studies. This has been true for photochemical oxidants and NO_2, as well as for SO_2 and particles (NAS 1975).

Despite all the limitations of the 21 studies listed in the Growth and Development table (and they are after all, only a small part of the research that has been consummated), and despite all the criticisms that can be levelled at the procedures employed and findings reported, and despite all the problems of extrapolation, surely--it would seem--that the concept of prevention which is so thoroughly embedded in pediatric culture should act as a motivating force sufficient to launch a program which would attempt either to affirm

or refute the relevance of these overseas investigations to American children.

Cancer, Mutations, and Malformations

There is substantial evidence linking polluted community air with increased risk from cancer, particularly cancer of the lungs--and a number of chemicals, metals and metallic compounds, fibers, and radionucleides have been incriminated. Among the chemicals are the polycyclic aromatic and the azaheterocyclic hydrocarbons; among the metals and metallic compounds, nickel, chromates, beryllium, arsenic, iron; and the major fiber, asbestos.

Difficulties in identifying specific airborne pollutants as carcinogenic for children, or as mutagenic or teratogenic are too numerous to list: the customary time lag between insult and reaction in the case of malignancy is, of course, a major factor; and the almost infinite variables the comprise the genetic picture and malformations as well seriously complicate the problem.

However, cancer is the leading disease cause of death among children. And illnesses of genetic origin account for a high percentage--something on the order of perhaps 40%--of children admitted to pediatric hosptial care. Just how much or how little of this mortality and morbidity can be attributed to environmental pollutants

is undetermined.

Cancer and Occupational Exposure of Fathers

Particularly from the field of occupational medicine, clues are beginning to surface which show a strong relationship between certain pollutants and cancer in children. For example, Selikoff (1975) believes:

> The spread of occupational illness [specifically pleural mesothelioma, a tumor of the pleura] to families of workers [primarily in asbestos factories] is a general rule. It's not a curiosity.

There are thousands of other workers exposed to contamination from asbestos, for it is used in the manufacture of innumerable products such as air-conditioning ducts, brake linings, fireproof insulation, etc., and in shipyards and the construction industry. Persons living near such factories or shipyards are also endangered. (Many asbestos-containing products can also pollute the air in homes, schools, and other buildings, as discussed in Chapter III.)

A research group (Fabia and Thuy 1974) at Laval University Medical School, Canada, recently checked the incidence of cancer among children whose fathers were employed as auto mechanics, miners, machinists, and painters. Comparing the family backgrounds of almost 400 children who died of malignancy before age 5 and those of nearly

800 others born at the same time, they found a two to one excess of those engaged in hydrocarbon-related occupations. Cases and controls did not differ with regard to place of residence of parents or mean paternal age.

How exposure of the father to hydrocarbons could lead to a greater risk of malignant disease among his offspring is not known. The researchers ask,

> Could it be because the child is himself exposed to the repeated or continued presence of certain items in the household or prolonged contact with his father? Could it be the result of a direct effect of some hydrocarbon contained in petrol and oil on spermatogenesis--the carcinogenetic defect would then be transmitted to the child?

Along this same line, a study made by National Institute of Occupational Safety and Health (NIOSH), CDC, and University of North Carolina School of Public Health (1976), just released, points to a doubled rate of miscarriages and stillbirths in wives of men working in vinyl chloride monomer plants as compared with those in rubber and polyvinyl chloride plants. Apparent cause: sperm cell damage in the female through direct vinyl chloride monomer exposure.

Mutagens and Teratogens

The data base for mutagenesis and polluted community air is on much less firm ground, but likely candidates among airborne noxae are certain oxidants, including ozone, also SO_2, nitrogen oxides, and photochemically

activated hydrocarbons; as well as benzene, organic peroxides, epoxides, lactones, and fungicides.

Only a very small percentage (perhaps 2-3%) of all congenital malformations in humans can be attributed to drugs or other chemicals in the environment. Among the known or suspected teratogens are organic mercury, polychlorinated biphenyls (PBC's), lead and certain pesticides.

Transplacental Hazards

Increasing attention is now being paid by fetologists, neonatologists, and geneticists to the role of transplacental hazards. It has been shown that fetal exposure to a chemical carcinogen facilitates the effect of postnatal contact with the same or a different carcinogen. This finding suggests that a person's lifetime risk of developing cancer may be conditioned by prenatal chemical exposure. Fraumeni of the National Cancer Institute (1974) says:

> The same chemical that causes abortion in the early stages may produce malformations during organ development and neoplasia when exposed later in pregnancy.

There seems to be a close relation between the processes of carcinogenesis and mutagenesis. For example, of more than 170 chemicals tested by Ames and his associates (1976) that are known to cause cancer

in animals, 90% were found to cause mutations in bacteria; whereas few of the 109 non-carcinogens had such an effect. Hopefully new insights will follow reasonably fast.

Many prenatally determined diseases, it is believed, may in fact ultimately be traced to the influence of multiple agents in the environment, and to complex interactions between these agents and susceptibility factors in the host. A sense of urgency to determine more about fetal physiology and pathology in relation to environmental pollution has been expressed (Scanlon 1972). The need for a comprehensive national surveillance and registration system for mutations and birth defects (as now exists in England) has also been voiced (Epstein 1973, AAP 1974), but so far has not been implemented.

Also voiced is the urgent need for establishing priorities for testing perhaps tens of thousands of untested chemicals and their mammalian metabolites (which serve to activate innocuous chemicals into highly toxic derivatives). For "extensive human exposure" to environmental chemicals is viewed by many geneticists as a "serious threat to the future of mankind as we know it" (de Serres 1976).

CHAPTER V

ARE PRACTICING PEDIATRICIANS CONCERNED ABOUT POLLUTED AIR?

Pediatricians are guardians of the health and well-being of infants and children, but have shown little concern with respect to degraded air qualtiy. Was this a real or only an apparent lack of concern? If real, what was the root cause?

The motivation behind this exercise came from an interest of long standing over what was considered a lack of solicitude or anxiety on the part of many pediatricians concerning the effects of polluted air on their patients.

To determine how the pediatric community assesses the risk of air pollution as it may affect the young patients under their care--an attempt was made to gain some insights by two different approaches. In the first, a survey of pediatric literature was undertaken to determine what information had been disseminated through such media. In the second, personal interviews with active practitioners were undertaken to probe various aspects of their knowledge of the subject, their reaction to new data when made available, their expressions of attitude,

and their professional activity, including instructions to patients for coping with air pollution.

Air Pollution in the Pediatric Literature

The literature search covered both pediatric journals and pediatric textbooks, and involved checking a number of subject heads--such as environmental hazards, smoking, poisons, respiratory tract illnesses, allergy, asthma, etc.--besides the obvious one of air pollution. The journal search started with the 4 publications devoted to diseases of children which were extant in 1950, and continued with 3 others which commenced publication in the 50's and 60's. It terminated with 1974 issues. The Connecticut State Medical Journal was also checked for the past ten years (since this is the State in which the interviews, about to be described, were to be conducted). In all, over 90 journal years* were examined. Besides the journals, a dozen specialty textbooks published since 1968 dealing with pediatric practice, pediatric diagnosis, pediatric therapy, and pediatric management were examined under the same headings.

The yield from the search of the literature was

*Eight different journals (7 pediatric) were examined for periods ranging from 8 to 25 years, culminating in a total of more than 90.

minimal. Except for brief sporadic mentions, the total input of information regarding the impact of community airborne toxic elements available in pediatric journals was confined to 5 articles or conference proceedings: the first 4 of those appeared in the official publication of the American Academy of Pediatrics (AAP), Pediatrics, as follows:

AAP's position paper (1970) on "Pediatric Aspects of Air Pollution";

a government report (National Air Pollution Association--Pearlman et al.--1971) on "Nitrogen Dioxide and Lower Respiratory Illnesses";

two AAP conference proceedings and presented in special supplements to the official journal: one held in 1966 on Radio-active Fallout, the other in 1973 on Chemical Pollutants, described below;

the fifth publication--a clinical review (Scanlon, 1972) on trace element hazards to the human fetus--appeared in Clinical Pediatrics.

Only two textbooks referred to air pollution, and in just one of these was the reference of any moment: this was a chapter devoted to the subject of its influence in cases of allergy (Girsh 1973).

Also in one annual meeting--1971--AAP held two symposia relating to environmental hazards: one on air and water quality; the other on mists, aerosols and oxygen. And at the 1974 meeting, before the Section on Community Medicine, EPA's Knelson (1974b) presented a paper on "Environmental Hazards for Children."

In addition to these publications on community air pollution, AAP (1969) presented a position paper on "Smoking and Children: A Pediatric Viewpoint."

By contrast with the dearth of material on air pollutants, other environmental problems were not ignored. There were close to 300 mentions* of poisonings and accidents plus a special supplement in 1969 in AAP's official journal devoted to "Childhood Injuries," and consisting of a great number of articles. Also there were some 70-odd articles dealing more or less exclusively with lead poisoning.

What Might Have Been Learned from Pediatric Literature

What might the most motivated pediatrician have learned through his professional journals? From the 1969 position paper on smoking, he'd have learned of the

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*There included original contributed articles, editorials, conference reports, case reports, commentaries, clinical notes, etc.

deleterious impact on children, and of the importance of informing youngsters of the hazards and of trying to keep them from taking up the habit.

From the 1970 position paper of AAP's Committee on Environmental Hazards (CEH), he would have learned that the majority of reports on the pediatric aspects of community air pollution had appeared in countries outside the U.S., and that a host of pollutants (such as sulfur dioxide, hydrocarbons, particles, oxidants, carbon monoxide, hydrogen sulfide, lead, arsenic, etc.) had been incriminated because of their effects on children's respiratory tracts, hematopoietic systems, athletic performance, growth, etc.

From the 1971 report on NO_2--the only study he would have seen dealing with American children--the most motivated pediatrician would have learned that youngsters in Chattanooga, Tennessee, experienced lower respiratory tract problems if they had lived in that area for a few years.

Again, he would have learned something--but little about American children--from the May 1974 Supplement to Pediatrics. This supplement covered the Proceedings of a conference (held in June 1973) devoted to "The Susceptibility of the Fetus and Child to Chemical Pollutants"

under the auspices of AAP's CEH, National Institute of Child Health and Development (NICHD), and National Institute of Environmental Health Sciences (NIEHS). In this supplement, several articles referred to air pollution, but presented little with respect to its effects on children; and one by a British scientist dealt exclusively with air pollution.

From the Britisher's remarks, the pediatrician would have read that levels of smoke and SO_2 are "much higher" in England than in the U.S., and that--though ventilatory function, and bronchitis, pneumonia, cough, phelgm production, and chest illnesses are affected by air pollution--"one of the great problems in the field of air pollution and respiratory disease in children is to determine whether air pollution really does have an effect." He would have observed also that there is a great deal of consideration being given the problems of carcinogens, mutagens, and teratogens (not necessarily air-borne). And he would have heard of the Alert Practitioner Program just instituted at three university centers in the U.S. essentially to check on the mutagenic-inducing potential of chemicals.

If a pediatrician had read each article word for word, he would have seen that one member (Finberg) of AAP's CEH strongly believes that, though the relationship may

not be a simple one of cause and effect, whatever else is going on, air pollution aggravates asthma and probably almost all respiratory disease. He would also have read that, in this pediatrician's view, asthma has been increasing exponentially at the same time that air pollution has worsened, and he would have read that a Texas pediatrician felt his colleagues were concerned about air pollution.

From another pediatric journal--not AAP's official publication--he would have learned that the Chairman of AAP's Massachusetts Chapter of the CEH was "disheartened" by the "low frequency of response" by the 350 Fellows of AAP in the Bay state in a survey on environmental problems.

The least motivated pediatrician (in terms of being the least well-read in his own literature) would have gained virtually no information on the subject of risk from air pollution to pediatric health.

What Active Practitioners Think, Feel, and Do

Following upon this search of the professional literature, the next move was to attempt to determine by personal interviews how active practitioners of pediatrics view air pollution in relation to the infants and children

under their care. It was felt that a sample of 40 such men and women--representing approximately 9.5% of the total number of pediatricians, and perhaps 12% of the active practitioners (as opposed to full-time university- or hospital- or institution-based pediatricians) in Connecticut--would be a manageable group, and should provide a reasonably accurate view of Connecticut pediatricians' perception of risk. Both a "Dirty Air" area and a "Clean Air" area were chosen, and this selection was made upon the advice of the Principal Air Pollution Engineer (Gregory Wight) of the Department of Environmental Protection of the State of Connecticut. (The cities and towns for each area are listed in Appendix D-2.) In Connecticut, two pollutants compose the index of pollution: sulfur dioxide and particulates. In the Dirty Air area, the concentration of SO_2 is approximately twice that in the Clean Air area, and that of particulates approximately 25% higher.

Interview Procedure

Names of pediatricians were selected in alphabetical order from the Yellow pages of the telephone directory and checked against a list of licensed pediatricians supplied by the State Department of Health. As the list of active practicing pediatricians for a city or town was

exhausted, additional towns for the Dirty Air and Clean Air were supplied by the Principal Air Pollution Engineer above referred to.

The following steps were then taken:

(1) a tentative questionnaire was developed and tested (Appendix D-1 is a copy of final questionnaire);

(2) a letter was sent to a dozen of the 40 MD's in the first city in the Dirty Air area, requesting a 20-minute interview (see Appendix D-2 for copy);

(3) a phone call was made several days later to set up a definite appointment (in most cases several phone calls were required);

(4) interviews were completed as expeditiously as possible;

(5) additional letters were dispatched as time permitted, and the process repeated until 40 interviews with active practitioners had been completed.

In all, letters were sent to 32 pediatricians in the Dirty Air area, and to 31 in the Clean Air area. Twelve clinicians in the Dirty Air area and 11 in the Clean Air area asked to be excused from the interview, most pleading either time pressures or ignorance about air pollution. (If there is a bias in the sample, the latter would suggest it is in the direction of higher

knowledge or concern with air pollution.) A total of 3 months and 3 thousand miles were involved in the interview process.

Public health officers in the cities and towns in both areas were also interviewed; and some but not all of the same questions asked. Their views will be discussed briefly below.

Sociologic Profile

All of the interviewees gave generously of their time, graciously fitting the interviews into extremely crowded schedules--a number giving up lunch hours and two coming in on their days off to confer.

Were it possible to judge the busier group, it would seem that those in the Clean Air area had perhaps the more hectic schedules. An age comparison shows them to be slightly younger in the Clean Air area (average 45.8 years) as against 53.5 years for the Dirty Air area. Average for the combined group is 49.5 years. More than twice as many are engaged in solo practice in the Clean Air area, in contradistinction to group, partnership, or prepaid plan practice in the Dirty Air area. There were only three women interviewed. Except as otherwise noted, neither age, nor location, nor type of practice seemed to influence responses.

Knowledge and Its Source

In responding to the question "Do you believe any of the conditions you treat are caused or aggravated by air pollution?", a heavy preponderance of the 40 interviewees (Table 6) felt that allergy or respiratory tract illnesses (RTI) or both are aggravated (to some degree at least) by polluted community or indoor air.

TABLE 6

VIEWS RE IMPACT OF AIR POLLUTION IN 2 AREAS
(expressed in percentages of interviewees)

	Dirty Air		Clean Air	
None or Minimal Effect	35		20	
Positive Effect in Allergy	25		30	
Positive Effect in RTI	10	65	30	80
Positive Effect in both Asthma & RTI	30		20	

As is obvious, there was not too much difference between the views of MD's in the two areas, except for the greater number in the Dirty Air area who saw a relationship between respiratory disease and air pollution, and for the source of pollution--with 6 MD's from the Clean Air area and 1 from the Dirty Air area specifying indoor (as opposed to outdoor) air as the major source

of exposure. Interviewees, on being shown a statement by an EPA physician about understating the true impact of air pollution, were asked whether they agreed or disagreed: over 75% agreed.

Roughly half of all the interviewees (as will be seen from Table 7) learned about air pollution and its impact on health/disease at medical school (often in connection with industrial medicine problems), or during professional training (as with an allergist), or as a result of personal experience (in World War II), or because of early residence in a heavily polluted area. More learned about polluted air through the lay press than their own professional journals--in fact, only three interviewees mentioned professional journals as a source of information.

TABLE 7

SOURCE OF INFORMATION RE AIR POLLUTION IN 2 AREAS
(expressed in percentages of interviewees)

	Dirty	Clean
Med.Schl/Prof.Train/Experience	45	55
Prof. Jnls.	10	5
Lay Press/Lay Activities	25	10
Combination (Lay & Prof.)	5	15
Don't Know	15	15

Attitude Toward Recent Evidence

To determine this attitude toward recent evidence, several exhibits were shown. The first dealt with findings relating to the effects of pollution on respiratory tract illness in different New York City communities. In showing this exhibit (an abstract together with complete report, Appendix D-1.1), the following with statement was made and the following question asked, "Here is a brief reference to recently published research by an EPA physician. Do you feel that his findings are applicable to your area?" The next exhibit (a letter and a professional journal article, Appendix D-1.2) dealt with the subject of mutagens; the following statement was made and the following question asked: "Air pollution has been statistically associated with congenital malformations, as you'll note from this excerpt from the studies of a UPenn scientist. Do you consider this valid?" And the third exhibit (a brief newspaper reference plus the original publication in Lancet, Appendix C-1.3) was shown with the following statement and question, "Here's reference to a recent British study concerning the dangers of smoking in the presence of infants. What's your reaction to this?"

With respect to the relevance of the EPA report to their area, considerable and logical disparity

appeared between the two groups, with 85% of the Clean Air area MD's believing this had minimal relevance for their practice and 15% some relevance, whereas half of those in the Dirty Air area felt it had minimal relevance and 50% some relevance.

While more than half (55%) of all pediatricians found the relationship between malformations and air pollution a possibility, the difference between Dirty and Clean Air area pediatricians was striking: 35% of the Dirty Air area MD's considered it a possibility; 75% in the Clean Air area. One reason for this difference may be because there are more younger MD's in the Clean area with more recent training and more knowledge of the relatively new science of fetology and neonatology.

In connection with the deleterious impact of smoking in the home, 60% of all MD's found that this Lancet report either "made good sense," or was "very interesting," or was "something with which they could agree wholeheartedly." Two had already seen it. The balance (40%) took the opposite view or had no clinical judgment: they doubted the findings, considered them alarmist, or had not found this to be the case in their practice. (A few of those who took the opposite view were themselves smokers.)

It would seem no exaggeration to say that, in

general, the interviewees reacted reasonably positively to the information shown them. While a few questioned methodology, and some asserted that statistics could be manipulated, and most wanted more time for a fuller perusal of the data, nevertheless the material had an interest for them. One or two wanted to read further, and took note of the Lancet reference. Interest in additional data was evidenced by the fact that over 90% expressed a desire to receive the AMA Guide* offered, and subsequently either handed or mailed to them. (Unfortunately, this Guide--Appendix D-3--contains little information about the impact of air pollution on the health of infants and children.)

Action Taken with Respect to Air Pollution

On a societal level, little has been done by the pediatrician about community air quality, although 11 interviewees have taken some positive local action. For example, 2 pediatricians in one town served on land planning and tree planting committees. Another is a member of his area health planning council, and advocates a strong emphasis on environmental considerations, including air quality. Two have appeared before appropriate municipal officials with respect to lead poisoning.

*The Physician's Guide to Air Pollution (1973).

Several have contributed to Lung Associations. And one has written to his state legislators in an effort to achieve some restriction against smoking in restaurants. Another is checking out several cases of malformations occurring in a community where he suspects polluted air as the causative agent. And, finally, one pediatrician works regularly with elementary grade youngsters in an effort to keep them from taking up the smoking habit.

Organizationally, pediatricians receive little support for such efforts, as evidenced by the failure of the Connecticut Chapter of AAP to follow the lead of some dozen State Chapters in setting up Committees on Environmental Hazards.

More action has been taken in connection with indoor pollution in the form of advice to patients. A heavy proportion (70%) of all pediatricians routinely (or "when appropriate"--for asthmatics and children with chronic respiratory conditions) advise parents to eliminate smoking at home. Other recommendations include proper ventilation for heating and cooking equipment, adequate precautions against polluted dust and/or allergenic or irritant particulates, and against aerosols. More Clean Air area MD's (65%) give advice concerning indoor pollution than Dirty Air area MD's (45%).

How Concerned Are Connecticut Pediatricians?

A measure of concern was a central focus of the interviews. Several parameters--admittedly very crude measurements--were used to assess concern in terms of either negative or positive response to the following: (1) knowledge and belief about (a) air pollution as either a cause or aggravating influence in connection with any of the conditions pediatricians treat, and (b) whether the effects from polluted air are understated or exaggerated; (2) attitude toward each of the three exhibits shown (as discussed above); and (3) behavior in terms of (a) community action, (b) advice given to patients about tobacco smoking, and (c) other steps recommended to patients for counteracting contaminated indoor air. Included also were (4) unsolicited observations, made not in response to any question in particular, but offered spontaneously as a reaction to the general problem of air pollution.

Typical of negative spontaneous observations were these:

> Unless I see a wheezing child dragged in off the street during an inversion, I wouldn't believe it.
>
> I'm more impressed by the germ theory.
>
> Man-made dirty air or Nature-made pollens, what's the difference?

I can honestly say I don't know of a single case of respiratory tract illness that can be attributed to air pollution.

Typical of positive spontaneous observations were these:

I don't trust "lots of the figures" about no cause for worry.

I believe all disease is environment-related.

People aren't prepared to bite the bullet for clean air.

A teenager here [one of the cleanest towns in the State] was lead-poisoned as a result of baby-sitting at her father's gas station.

I see "more and more" emphysema among children and childhood bronchitis becoming both more severe and more chronic.

I know the damage air pollution can cause: I had Tokyo-Yokohama asthma during Army service in Japan.

In a steel mill town where I grew up, if smoke showed in the stacks, you knew you wouldn't feel as well, but you would have work.

I'd hate to be a toll collector.

Measures of Concern

Thus there are 4 measures of concern (Table 8).

Few of the individual pediatricians are wholly consistent in their negative or positive ratings. Nevertheless, it is possible to allot most of them by a composite rating to one or another group, as in Table 9.

TABLE 8

MEASURES OF CONCERN FOR AIR POLLUTION

			Negative	Positive
Knowledge and beliefs, av.			22.5	77.5
	Neg.	Pos.		
2 factors	22.5	77.5		
	22.5	77.5		
Attitude toward proferred information, av.			54.8	45.2
3 exhibits	67.5	32.5		
	45.0	55.0		
	40.0	60.0		
Behavior, av.			49.2	50.8
3 factors	72.5	27.5		
	30.0	70.0		
	45.0	55.0		
Spontaneous observations			35.0	65.0

TABLE 9

PEDIATRICIAN RATINGS OF CONCERN

	# of Pediatricians	% of Pediatricians
Predominantly Negative Concern	12	30
Mixed	5	12.5
Predominantly Positive Concern	23	57.5

The two extreme groups might be further

characterized by their archetypal views reminiscent of epidemiological or public health philosophies.

"Mind-The-Iceberg" Type

The concerned group with strong positive feelings are "Mind-the-Iceberg" types (MTI). A MTI'er (Figure 4) is one who, on seeing the tip of the iceberg, is sensitive to, and concerned about, the dangers that may lurk below. An MTI'er is one who rates high on at least 3, if not all 4, of the measures of concern. He believes that polluted air has a positive impact on some of the cases of respiratory tract disease that he treats (and possibly other conditions as well), and that we are probably more prone to understate rather than exaggerate its adverse effects. He is interested in or receptive to (if not already acquainted with) the evidence shown him. An MTI'er may or may not have taken positive community action; he usually gives advice with respect to coping procedures for indoor pollution. Above all, his spontaneous observations about air pollution in general (and in response to the question about what an individual MD can do to upgrade community air quality) give evidence of concern--of solicitude or even anxiety about the adverse impact of airborne noxae.

Fig. 4. "Mind-the-Iceberg" type sensitive to dangers lurking below

"Count-The-Bodies" Type

The unconcerned pediatricians with negative feelings are akin to the "Count-the-Bodies" (CTB) type people. A CTB'er (Figure 5) stands at the opposite end of the pole philosophically from the MTI'er. He demands incontrovertible proof of damage before taking a position. He reacts negatively to at least 3 out of 4 of the measures of concern. He largely discounts the relationship between polluted air and the conditions he treats, and feels that its ill effects are probably exaggerated. He is not particularly interested in or impressed by the evidence shown him. He has taken no community action, though he may give advice to allergic (and in rare instances) to chronic respiratory cases regarding precautions to be taken to avoid problems presented by unfavorable (but not necessarily polluted) indoor air. And, by his unsolicited remarks and general attitude during the interview, shows little or no concern about ambient air quality. Those with mixed concern have beliefs similar to the MTI'ers, but their attitude is a bit more skeptical, and they have taken little or no action at the community level.

This division reflects the wider division in the scientific community related to a broad range of environmental problems, and thus there is little surprise in

Fig. 5. "Count-the-Bodies" type
demands incontrovertible proof of damage

the classification. However, what is probably more significant for the majority of pediatricians concerned with air pollution is that there is so little in their literature or in their organization to support their interest.

Interviews with Public Health Officers

In addition to the 40 pediatricians interviewed, 3 health directors (2 full-time and 1 part-time) were also interviewed: of these, 2 were MD's and 1 a registered sanitarian and master's degree candidate. Of the 2 MD directors, one was a pediatrician, the other a general practitioner. In addition, 3 maternal and child health (MCH) personnel were seen: 1 serves at the State, and 2 at the City level.

Because of the small numbers involved, and because of their divergent backgrounds as well as their divergent duties--school physical examinations of children, maternal and child health clinic services, medical attention at athletic events, inspection of food service establishments, of septic tanks, and the like--it is difficult to come up with any meaningful conclusions as to their concern about air pollution and children's health.

Among public health professionals in general, such dangers as may arise from air pollution do not rank high among their list of problems demanding solution.

Among the poor, overcrowding, poor housing, improper nutrition, inadequate prenatal care, lead poisoning (but not necessarily arising from airborne lead), etc., are considered much more seriously aggravating conditions. Among the middle class, improper nutrition and dental caries would appear to have top billing as deterrents to optimal child health.

One pediatric public health officer had attended a meeting called by the Department of Environmental Protection (DEP), but didn't feel that she had either learned or accomplished much because the entire session was devoted to a discussion of mass transportation. Though a dozen services are offered by the MCH section of the State Department of Health (including information of various sorts, such as accident prevention and child abuse), nothing regarding air pollution is offered.

Two of the 6 health officers expressed concern about air pollution from tobacco smoke in the home and in schools. One who felt that "99% of respiratory troubles come from tobacco smoke" believed that the only way to stop smoking in schools was by hiring only nonsmoking teachers, and by abolishing special smoking rooms in schools. One MCH specialist said that because so many of our free clinic mothers are attempting to cope with "earth-shattering" problems and seem devoid of hope, "I

can't bring myself to tell them to give up cigarettes--which seem to represent one of their few pleasures--though it disturbs me to think of their blowing smoke in their baby's face." Another MCH'er asked whether cigarette smoke was considered a form of air pollution; he had never heard that smoking in the home could harm an infant.

Commentary

Is the hazard of air pollution to children greater or less than the risks assessed, or is it different (Kates 1975 suggests these three possibilities)? This question cannot be answered with a high degree of confidence now, for assessment research is ongoing, and more time is required for statistical evidence which is considered fully or adequately confirmatory, or the opposite. What perhaps can be answered now is whether the lack of concern in the pediatric community as a whole concerning the impact of air pollution on infants' and children's health is real or apparent.

If judgment were made solely on the yield from the literature research (described above), the only answer possible is that the lack of concern is--for the most part--real. If one measures concern about an environmental hazard by the number of published references to it (recalling the approximately 300 articles or notes

about poisons in the pediatric literature mentioned above), surely it is no exaggeration to state that air pollution as a hazard is assessed as a low risk.

However, based on an analysis of the personal interviews, concern is real on the part of a little more than half of the practicing pediatricians. It is a concern which is sporadic, not cohesive, not organized, and not well articulated.

Perhaps the most critical question is the second question raised in the introduction to this chapter: If the lack of concern is real, what is the root cause? This question might now better be revised to, What is the cause of this lack of concern on the part of pediatric official-dom? For, if concern exists among a sizeable portion of the toilers in the field--the practicing pediatricians (as just described)--why isn't this concern shared by the "leaders"?

It is suggested that there are probably several major causes for this lack of official concern. The first is AAP's role, and particularly that of the national Committee on Environmental Hazards. Too many interviewees to be ignored said that they looked to their leaders--the AAP--for guidance, but found little ("Why no brochures on this problem from AAP as on so many other problems," one asked? "I've seen nothing in Pediatrics about this,"

another said. "We don't know anything about air pollution; we need a book," observed a third).

The second is EPA's role and its failure (for the most part) to communicate the findings from its community studies to pediatricians. Lesser roles--in the sense that practicing pediatricians are not involved in any direct way with them--are played by NICHD and NIEHS. Lesser but influential roles as far as doctors now in practice are concerned are also played by the medical schools. Let us look at each of these in turn.

AAP's CEH has taken what amounts to a "no-position" stance--a stance which serves as a powerful deterrent to action (Crenson 1971). For example, the Committee has failed to update its October 1970 position paper, and therefore has done nothing to remedy the "relative lack of serious consideration of the problem [of air pollution] in this country" which this paper referred to. While it sponsored the 1973 Conference on "The Susceptibility of the Fetus and Child to Chemical Pollutants," in co-operation with NICHD and NIEHS (Proceedings which were published in the May 1974 Supplement to the official journal, as mentioned above), very little information on the effects of air pollution in American children was presented. Furthermore, only 35 pediatricians were invited to attend this conference, so the

on-site exposure--and apparently the spin-off also--was minimal.

Again, while the nation's chairman of CEH presented a summary of current information on air pollution and the illnesses of children at an AMA Conference (San Francisco, December 1974) on air pollution, his talk was heard by just over 100 MD's (the number of pediatricians attending being unknown).

Let us look briefly now at EPA's role. Only one communication by EPA (actually its predecessor National Air Pollution Control Association) has appeared in Pediatrics. This discussed its study in Chattanooga, Tennessee, with respect to pollution damage from NO_2, reported in 1971 (above referred to). While several of EPA's other community study reports have subsequently appeared in professional environmental health and public health journals and in their own government-sponsored reports, pediatricians in general haven't been privy to the results of EPA's community studies.

What about NICHD and NIEHS and their role? In the foreword to the Proceedings of the 1973 Conference on "The Susceptibility of the Fetus and Child to Chemical Pollutants," Miller refers to the "serious problem" uncovered by the Conference: No federal health agency has the responsibility for research into the special exposures

and susceptibility of the fetus and child. There are scientists, he said, who know about environmental effects but not about children's health, and pediatricians who know about children, but haven't given much thought to environmental effects. Miller believes that:

> The many observations described at the meeting can be used by AAP, various health agencies, individual investigators and practitioners as springboards for research or civic action.

(One wonders whether AAP has plans for any research or civic action concerning air pollution problems.)

Next, for a moment, let us think about medical schools and what their role has been with respect to the health effects of air pollution. In his summary of this same conference, after commenting on the fact that the physician is "badly educated" in regard to the whole area of environmental and occupational health, Case Western University's Dean (Robbins 1974) had this observation:

> This situation requires attention at all levels of our educational system, and not just in medical school or in continuing education for pediatricians. In general the medical schools are doing a miserable job on this.

Along this same line, several interviewees faulted their medical school education (where, though they first heard about air pollution under industrial conditions, they said, virtually no extrapolation from

industrial to community exposure was made), or their later professional training, perhaps in allergy (where again virtually no extrapolation between allergic and nonallergic children was proferred). And many interviewees observed that, though they had been indoctrinated in the concept of preventive pediatrics, much of their practice was actually after the fact, and (except for immunizations) their time was spent largely in treatment.

Perhaps one reason for this lack of attention to environmental problems in medical schools is the fact that certain university-based pediatricians without private practice experience may be more concerned with research into esoteric conditions. (Children have always been subject to respiratory ills, what's new or exciting or important about this?) Another version may be that, because they feel there's inadequate hard data, they can't build a curriculum around soft data.

If medical men at the university level have appeared to show little concern for the deleterious impact of air pollution, the question arises whether this can be attributed to the fact that environmental considerations have up until only very recently been considered the sole province of public health professionals. And, if now environmental matters have in a certain sense become everyone's business, are they thus no one's

responsibility?

Still another question might be asked: Does the medical profession as a group shy away from concern over air quality because its control is outside its province; this belongs in the sociopolitical arena? (One reason given by a member of AAP's national CEH for pediatricians' failure to speak out about degraded air quality is that, as a rule, doctors are disinclined to "rock the boat.") On this point, interviewees voiced such views as: "The solution [to the air pollution problem] lies outside of pediatrics in the social realm"; "It's a responsibility of public health people or of engineers"; "Clean air is a function of good government"; "Who can stand up to the industrialists?"

Whatever the reason for the failure of pediatric society leaders and academicians to become more involved with, and more articulate about, the deleterious effects of pollution, it is suggested that the concern which exists among a considerable segment of active practitioners who were interviewed should command greater consideration than it has so far received. Since pediatrics--of all branches of medicine--has long been distinguished by its dedication to the principle of prevention, and since the consensus seems to be that dirty air is either causing or aggravating a certain percentage of the most common acute

conditions of childhood, and perhaps some of the chronic conditions as well--as discussed at some length in the preceding chapter--pediatric officialdom should be according the problem higher priority. Suggestions as to how more effective and cohesive protective action might be consummated by various groups concerned with environmental matters and with child health care form the subject of the following chapter.

CHAPTER VI

THE ENVIRONMENT AND PREVENTIVE PEDIATRICS

Benefits of Updating the Prevention Concept

"From the very beginning," said the American Academy of Pediatrics' (AAP's) incoming President in 1969, "physicians caring for children have been their advocates before society." Indeed, Hugh Thompson added, "our Academy was founded . . . to assure a forum for this sort of advocacy." "Inevitably," he felt, "pediatricians will be counted among the visionary social planners."

That same year, in accepting the Abraham Jacobi award, another pediatrician (Waldo E. Nelson) painted a quite different picture:

> In pediatric practice, much lip service is given to preventive medicine, but beyond active immunization procedures for certain infectious diseases, one may wonder to what extent the real potential has been achieved.

Both of these observations, it seems, have a special significance today. In view of the special hazards youngsters face from an air-polluted environment--delineated in the foregoing sections of this presentation--never before have they needed advocates before society more than now. And, by the same token, never

before have pediatricians had a more auspicious opportunity to realize their full potential as practitioners of preventive medicine.

The primary reason is that: since most of the infectious diseases of childhood have been conquered or effectively controlled, environment-related illnesses assume a larger share of the conditions with which pediatricians are called upon to cope.

Consider for example acute respiratory ills and certain chronic conditions like asthma. Though such disorders constitute a heavy proportion of the practicing pediatrician's daily patient load, far too few are apparently aware of what proportion of these disorders is either caused or aggravated by air pollution. Risk estimates have been made, but not well communicated. Take another example, cancer, now the leading disease cause of death among children. What percentage of malignant tumors in childhood are environmentally linked, no one can say, but it is generally acknowledged that an overwhelming proportion (perhaps 60 to 90%) of all cases of cancer are caused by environmental chemicals, many airborne. Likewise, certain CNS dysfunctions are environment-related. Also certain hematologic disturbances, enzyme system alterations, etc.

Were pediatricians to become internists for a day

(in short to project as futurists might do--to see the health of their small patients as it will be, say, in the year 2000), would their patient load then consist of as many chronic adult bronchitics, of as many emphysematous cases, and of as many CNS and malignancy problems? If so, might they as internists not wonder how many of these disabilities could have been avoided or greatly ameliorated if truly preventive pediatrics prevailed?

For the sake of optimal health for children, it would appear eminently sounder to proceed on the basis of the "MTI" approach described earlier, i.e., that the dangers lying beneath the surfaced tip of our technology may prove greater and more protracted than those presently visible. For what may appear to be successful adaptation to acute pollution situations may over the long term prove harmful with respect to chronic sequelae. The National Academy of Science report (B. Goldstein 1975) puts it this way:

> If adaptation does exist, it is conceivable that a lessening in response will be observed during a period of gradually decreasing atmospheric concentration, but--once the new concentration has become stable and is maintained long enough for adaptive response to wear off--variations around the lower concentration might again produce acute responses.

Further, it is highly conceivable that the savings involved in terms of the physical and psychic disabilities avoided because of early preventive care may

far outweigh the costs of such care. We are reminded that death from the socioeconomic point of view is relatively unimportant compared to chronic disability (Robbins 1974).

As far as the pediatric profession itself is concerned, it would appear that the great advantage of an updated preventive pediatrics concept--with a strong social advocacy base--is that it would relieve clinicians of responsibility for curing, and not merely relieving, many ills for which they presently have no effective therapy; only symptomatic palliatives. Of tremendous significance, would be the weight and impact of their contribution in increasing awareness of the need for and desirability of healthier environmental conditions, and for intensifying the effort to achieve and maintain cleaner and more aesthetically acceptable (and thus possibly more mentally healthy) communities.

Thus, since most environment-related illnesses can't be effectively handled in professional isolation without controls over noxious chemicals, it would seem that preventive pediatrics must perforce extend its sights beyond treatment in the customary office or hospital setting to a consideration of conditions in the larger society. For the true unit of health is, of course, not the individual nor even the family, but the community. In the case of air pollution, with the transport of

pollutants and their conversion and transformation products in the atmosphere far from their emission sources, the community becomes a larger and larger entity. And, since air pollution control is not a scientific or technological problem alone but a social one as well, it is suggested that pediatricians--to exert a positive preventive influence in the decade ahead--must willy-nilly accept the challenge of interaction with the sociopolitical world, with which they (as well as their patients) are inexorably bound.

As an EPA pediatrician (Knelson 1974c) expressed this:

> I am convinced that they [physicians] are largely unaware of the pressures for and against better control of these hazards [environmental hazards in a technologically advanced society] to the health of their patients and I believe they are largely unaware of the role they can play in assuring better environmental control.

Also an epidemiologist (Griffin 1974) pleads as follows:

> I would call upon the clinical community to be a part of the action, and to contribute the wisdom and understanding they possess about the health of the public and the management of this most important problem [health effects of air pollution].

Principles and Parameters of an Update for Preventive Pediatrics

An updated pediatric prevention program for

protection against environmental hazards (and particularly for protection against air pollution hazards) discards certain obsolete notions--such as the single factor concept of disease, and the need for incontrovertible proof before assuming a positive stance that air pollution is bad for health. This implies that such a program embraces the multifactorial concept of disease. And that it proceeds on the principle that demonstration of safety is more an obligation of those who have rendered (or would render) air unclean rather than that completely substantiated proof of damage because of such action is an imposition on those mandated to protect the health and well-being of the public.

Updated preventive pediatrics thus is social advocacy based on several premises:

(1) that, while the aim of the 1970 Amendments to the Clean Air Act is to insure and conserve clean air, constant vigilance is required for its successful and timely implementation because of the ever-constant and all-powerful pressure exerted to weaken and/or delay enforcement of all its precautionary regulations;

(2) that the air quality standards established for certain pollutants are neither inviolate nor wholly acceptable, since they were set with inadequate dose-response-age-activity relationships; nor are they in any

sense complete, since all the contaminants and conversion products and synergistic factors have not yet been taken into account;

(3) that children are a unique and sizeable minority requiring special pollution protection--different from most adults (except for the elderly)--because of their particular susceptibilities to insults from airborne irritants and toxins, as discussed in Chapter I; and because these insults may have a far-reaching and long-lasting effect; and

(4) that long-term economic benefits incurred in tendering sensitive consideration to little people in their work (play) places with different breathing zones under different pollution concentrations may far outweigh the impact of short-term dislocations involved in effecting the cleanup.

Essentially preventive pediatric practitioners can perform several important and useful functions in their role as social advocates for clean air. The rubric of consciousness-raising might appropriately be applied to the over-all process, which assumes three different forms: information-giving, pressure-making, and example-setting.

Consciousness-raising may be effected by individual doctors acting alone or in professional groups; it may take

place at local, state, and federal levels.

To divert for a moment, one of the most crippling constraints to be surmounted (or worked through) in the updated preventive pediatrics program is the present failure of communication between the numerous groups involved either in the delivery of health care to embryos, fetuses, infants, and children, or in epidemiological, laboratory, or statistical research, or in medical school teaching, or in environmental protection or enforcement work. Surely, if we can fly to the moon, build a thousand-mile pipeline, and construct a supersonic transport, alert concerned pediatricians can forge a more effective channel of communication between all those involved in one way or another with the health and well-being of little people. Opportunities can be provided for more interaction and pooling of insights which can prove constructive, meaningful, and beneficial for the pediatric population.

For example, interprofessionally, a good start might be made were pediatricians to arrange for greater liaison not only with allergists and pulmonologists (for quite obvious reasons), but with fetologists, neonatologists, geneticists, oncologists, obstetricians, and family practitioners. Many of these latter six specialists have become somewhat sensitized to the evidence

beginning to surface concerning the traumatic influence of certain airborne pollutants, including those capable of crossing the placental barrier, or adversely affecting sperm cells, and thus inflicting damage to embryos and fetuses.

Let us look now specifically in turn at what and how concerned segments in the child care and environmental protection world can do to update pollution protection in the days ahead.

What Individual Pediatricians Can Do

As an information-giver, the pediatrician can function with individual patients or with clubs and associations of various types and with differing interests and responsibilities--ranging from parents' and teachers' associations (PTA's) and League of Women Voter (LWV) chapters, to legislative committees, to unorganized groups--all anxious to learn from those with real expertise about the ill effects (both short- and long-term) of specific air pollutants at present levels in their communities, particularly where children play and study (at home and at school, and outdoors as well as in). Anxious to learn also about practical coping procedures--such as selecting home or day care center or school sites as far removed as possible from industrial or incinerator

emission sources or heavily trafficked areas.

With reference to indoor pollution, subjects such as the following (also discussed in Chapter III), bear emphasis:

impact of smoking by parents (especially mothers) on the young, since sidestream smoke would seem to represent a greater hazard than that inhaled by the smoker);

importance of persuading teenagers to refrain from starting the smoking habit;

precautions with respect to the use of asbestos- and lead-containing items;

use of substitutes for aerosol preparations;

proper maintenance of heating and cooking equipment;

avoidance of improperly vented wood-burning fireplaces;

vigilance needed for escape of pollutants from attached garages.

A number of pediatricians have considered the type of information-giving that might prove helpful. For example, interviewees made these suggestions:

increase public awareness of the air pollution problem by placing signs in the office;

make "non-scary" pamphlets available;

caution mothers against carrying small children around parking lots;

tell parents that every youngster, on reaching age 16, shouldn't automatically expect to be given his own jalopy.

There is another way in which pediatricians can counsel individuals and groups to help in preventing some of the damage caused or aggravated by airborne noxae. As NAS (1975) pointed out, the possibility that nutritional or pharmaceutical agents can modify the response to air pollutants cannot be overlooked.

Included among these nutritional agents which are considered helpful are vitamins A, B, C, and E, and calcium. For example, vitamin E may protect vitamin A from destruction by air pollutants, while vitamin A directs the formation of healthy cells in the lining of the lung. Vitamin A is said to be essential for "steering" the differentiation process of epithelial cells toward formation of healthy, mucus-secreting tissue (De Luca 1970). Vitamin E can prevent the loss of the polyunsaturated fatty acids such as linoleic acid from the lung lining of animals exposed to ozone or nitrogen oxide, and in humans has produced much greater resistance to cellular damage (Menzel 1975). A Swiss physician (Blumer 1974) has found calcium edetate plus vitamins B and C helpful in patients exposed to lead and other automotive pollutants and experiencing headaches, fatigue, or digestive disorders.

As a pressure-maker, the pediatrician's primary target group is elected representatives and government officials. His secondary target: polluting industries

and polluting life styles. His methodology includes personal representation, letter-writing, public addresses, and the like. His most urgent consideration at the moment might comprise some of the following:

> maintenance of present air quality and emissions standards (primarily SO_2) by resistance toward efforts to degrade standards or delay enforcement;
>
> upholding the need for adequate funding for Departments of Environmental Protection and for EPA;
>
> standard setting for lead (and other dangerous trace metals);
>
> restrictions against smoking in public places, including schools;
>
> improvement in mass transportation, including de-emphasis on highway construction;
>
> provision for more bikeways, etc.

As for personal representation, Medical World News' Washington Bureau Chief recently wrote (12 Dec. 1975):

> Members of Congress get crusty as barnacles when they listen to the well-paid arguments of vested interest groups, but they are surprisingly affected by the strong feelings of individuals--particularly if articulately presented.

As an example-setter, it was interesting to observe that a number of the interviewees--in recognizing their role as individual polluters--also recognized the importance of their own behavior and personal life styles in their communities (if you're not part of the solution, then you're part of the problem). They volunteered that

they might take such steps as these to aid in reducing pollution:

drive only small cars or those with Diesel engines;

buy only lead-free gasoline;

burn as few leaves as possible;

recycle as much as possible;

refrain from smoking in public, especially in front of teenagers;

join groups which "push" the government for greater protection, and which aggressively further consumer versus industry interests.

What Organized Pediatrics Can Do

"Union gives strength." In the past, AAP has proved a powerful force, among other very worthy causes, in the drive to prevent accidents--primarily accidental poisonings--among children. In updating its prevention program to embrace pollution protection, AAP (and other organized pediatric and general medical groups) can also function in the same three ways as individual pediatricians, though--acting as an official group--their methodology may differ in some respects.

As information-givers, AAP has an eager audience among Academy Fellows sensitive to its output and thirsty (as mentioned in the previous chapter) for data relevant to the problems of their daily practice. In addition,

there are other beneficiaries (like PTA and LWV groups) of pediatric-oriented data, which--when released by the organization specifically responsible for child health care--would be highly valued and highly valuable. Additionally, those groups concerned with environmental protection which haven't yet thought specifically (or been informed about) children's special requirements would be greatly aided. Through such information dissemination AAP would not only help in increasing the number of individuals and groups with insights into the unique vulnerabilities of the young, but could succeed in enlisting the backing of such groups to supplement its own efforts in furthering the cause of greater pollution protection. (One way in which AAP might quickly expand its influence would be by beefing up--through its State chapters--the number of its present local Committees on Environmental Hazards.)

As pressure-makers, AAP can function in much the same way--but with the additional clout that numbers and official status impart--as individual pediatricians in support of present or improved air quality standards, and of opposition to any lowering of standards or delays in compliance, as well as for pollution monitoring and enforcement, and (very importantly) for increased funds for research. Dovetailing its activities wherever possible with those of other professional specialty groups as

discussed earlier (oncologists, obstetricians, and family physicians, and the like) would no doubt nave an additive if not a synergistic influence.

As example-setters, AAP can take the lead in initiating increased interchange and mutual support with government agencies with some responsibility for children's health and welfare and environmental protection, such as National Institute of Child Health and Development (NICHD), National Institute of Environmental Health Sciences (NIEHS), Department of Health Education, and Welfare (HEW), Center for Disease Control (CDC), Americar Public Health Association (APHA), Environmental Protection Agency (EPA), etc.

What the Research Community Can Do

While EPA has devoted a great deal of its community surveillance work on the impact of sulfur oxides and nitrogen oxides on the health of children, by and large epidemiologists engaged in air pollution investigations (as mentioned in Chapter I) have studied children not because of the special needs of the young but because of the conveniences they provided. (An exception is investigative work on low level lead poisoning, described in Chapter IV). However, interest in the unique susceptibilities of the young to chemical pollutants is surfacing--as evidenced at the 1973 meeting sponsored by AAP, NICHD and NIEHS--and hopefully more research effort will be devoted to the impact of airborne noxae at the

breathing zone levels of developing organisms at various stages of growth.

Most every concerned professional has a priority list of research needs; they are many and diverse, even though funds and trained personnel are limited. One at the top of most lists at the moment would probably include a determination of threshold limits for the sulfates. Another would be some consensus as the the CNS effects of low level lead. Also, further information with respect to the impact of other trace elements like cadmium, arsenic, selenium and nickel is important. Research on mutagens, carcinogens, and teratogens is, of course, of tremendous significance; so, too, is the role of pollutants in increasing susceptibility to infection, in altering enzyme systems, and affecting hematologic reactions and bone maturation, etc.

Hopefully the research community will aim toward achieving a closer relationship with the clinical community (and vice versa) to the end that, along with pursuit of answers to some of the long-term threats to health, reasonably quick answers can be provided for some of the more common everyday conditions aggravated or caused by dirty air. Hopefully also AAP can serve as a catalytic force to stimulate interest in research concentrated on the problems of the young.

What Medical Schools Can Do

The time seems more than ripe for a greater emphasis on the part of most medical schools for the condition of human health as it is affected not only now in today's polluted environment, but as it may be affected tomorrow without more surveillance and control of unhealthy chemical fallout. Schools and departments in large universities, other than medical, have long since organized programs devoted to exploration of a wide range of ecological issues: such schools and departments as veterinary medicine, law, forestry, land planning, and urban affairs, to name a few. Somehow, schools of medicine have by and large lagged behind, in part perhaps because public health people have for so long considered the environment their particular turf.

However, because of the significance of environment-related illnesses, the expertise of full-time teaching and research staffs supplemented by part-time clinical faculty of medical schools surely should be brought to bear on the development of appropriate curricular material to plug the present lacunae, and to help budding practitioners to learn how they can cope with some of the pollution-induced or aggravated conditions which will probably be experienced by youngsters (as well as oldsters) in the 1980's. Inter-disciplinary efforts along

with other appropriate schools and departments might prove helpful and stimulating to all parties.

What Environmental and Child-Oriented Groups Can Do

By learning more about the distinctions which separate children from most adults (other than the elderly)--thus rendering the young more vulnerable to deleterious sequelae from air pollutants--environmental groups will be able to add this significant minority to give further weight to their pleas for rigid adherence to present air quality standards, and for inclusion of other noxious elements (like lead) in the list of regulated pollutants. And child-oriented groups (like PTA and LWV) can, by learning more about environmental effects, appreciate the need for greater pollution protection for the pediatric population. If geriatrics-oriented groups join in--since de-development is akin to development in terms of immunocompetence--all would be gainers.

A number of the Connecticut pediatricians interviewed (as described in the preceding chapter) bemoaned the after-the-fact nature of their present practice. It seems probable that this need for therapeusis may become intensified in the years ahead without an update on the prevention concept which embraces an awareness of the need for more effective environmental control.

Anticipatory guidance not just for children but for the community would represent a tremendous contribution by pediatricians.

APPENDIX A

STUDIES OF VENTILATORY FUNCTION IN AMERICAN CHILDREN
(Partial List)

Author	Publication	Study Area	Study Population: No.	Age	Sex	Race	Soc-Ec.	Study Method	Pllt Msrs.	Exposure Range	Findings	Critic	Publication	View Expressed
Chapman	1974 AMA Conf.	Chrlstt NC(clnr Brmghm Ala (drtr)	7,997 (3,705 in Ch, 4,292 in B)	7-13	M/F	Wh/ Bl	NA	3 $FEV_{0.75}$ tests on socec & dmrphclly smlr groups dur schl yr 71-2 in 12 elem schls in Ch (clnr area) & 10 in B (drtr); mns outdoor tmps & xpsrs to air p dur test yr rcrdd as were estmts of past p xpsrs fm 1959	RSP (dia mg3.5u)	36.0-44.4	Fdngs "cnsstntly lo'st" in more pllted c.ty for all 8 age-, sex-, & race-spc.. subgr.ps, strngly indctng that xpsre to prtclte p xrtd dltrs effct. Xpsre to RSP incldng SS fm bgnng of life prnts imprmnt in $FEV_{0.75}$ in later chldhd, & SS xpsre for past svrl yrs prnts such imprmnts.	NAS	1975 Air qlty & Stnry Srce Progn Control.p.73	A no of xprmntl shtcmngs (involvng eqpmnt, prsnnl, & hi'r ed attnmnt of parnts plus more schl busing in Ch), but "the maj postv fining is cnsstncy of effct of air p."
Ferris	1970 A-Rvw RespDis 102: 591-606	Berlin NH-3 areas	692	7-8	M/F	Wh	All class	Msrmnts of plmnry fnctn dn in 4 prchl & 3 pblc schls in Jan (xcpt for 1 schl) & Je 1967 by svrl mthds: pk flow, FVC & FEV; ch mtchd for ht, age, & sex	SO_2 (μgSO_3/ 100cm^2/ day)	206-619	"Msrmnts of plmnry fnctn ... did show sgnfcnt dfrnce that cld hv bn due to air p." Fndngs "might indicte that the prtclte cncntrtn, as indctd by dustfall, is as impt, if not more so, than the cncntrtns."	NAS	1975 ibid., pp. 63-64	Crtcsm vldty of fndng bec worst schl (A) in trms of plmnry fnctn was of a lo'r socec class than most, xcpt for schl J in unpllted area; dfrnce bet these 2 not atstclly sgnfcnt.
									SP dustfall(tons/ mi^2/mo)	13.4-43.2				
Mostardi &Leonard	1974 Arch Envh Hlth 29:5 325-328	Brbrtn O(pltd) & Rvrw (clnr)	92 42 in B, 50 in R	teens	M	Wh/ Bl	NA	2 dfrnt stds: (1) 1970 amng jrchi stdnts, (2) 1973 amng smllr smple orgnl pop; 3 tsts plmnry fnctn (VC, $FEV_{1.0}$ & MMF) on all stdnts, & VO_2 max on 30 smlr in age, ht, wt, & socec status; yrly avrgs for pllt nts for pd 1959-73 rcrdd	SO_3 (mg/100 cm^2/day)	Yr 1972: 0.36-1.020	B group had atstclly sgnfcnt lo'r mn VC than R; also atstclly sgnfcnt lo'r VO_2; no dfrnce in $FEV_{1.0}$ & MMFT. "Since vals for ambt pllt nts are cnsstntly hi'r in B, it is cncldd that sch pllt nts shld be cnsidrd impt cntrbtry factrs in the imprmnt of cardiopimnry fnctns... Fndngs may indicte that there cld be sm prmnt imprmnts of cardiopimnry fnctns as a rslt of crtn air pllt nts."	NAS	1975 ibid., p. 65	"Potntl crtcsm" incldg rltvly sm no stdnts tstd, lack of prsntl data on cig smkng (tho qstn was askd), & cntmpry urb-rur dfrnce in use of nontbcco cigs. "This stdy tnds to sprt an assctn bet a dcrmnt in chldhd plmnry fnctn & air p slightly stv the crnt US stndrds for TSP & SO_2."
									Dustfall (tons/ mi^2/mo)	9.20-25.00				
									TSP (μg/ m^3)/day	79.95-100.12				
Shy et al.	1970 JAPCA 20:539-545 1973 Arch Envh 27: 124-128	Chtnga Tenn 2 pllted areas, 2 cntrl	987	7-8	M/F	Wh	Mid Lo'r schls in hi pltcl area	$FEV_{0.75}$ prfrmd once wkly & midfm Nov 68-Mar 69 in 12	NO_2	86-166	Vntltry prfrmnce of 2nd gr schlch in hi NO_2 xpsre area was sgnfcntly lo'r than in cntrl area. The data sgstd that vntltry prfrmnce was alvrsly affctd only when an NO_2 thrshld was xcdd but that abv this thrshld no frthr imprmnt cld be dtctd.	Griffin	1974 AMA Conf.	Prblms & rlblty of mntrng instrmnts, smltns prsnce of oth pllnts, & swht less than straight-line dose-rspnse rltnshp.
									SS	1.6-5.8				
									SP	9.8-11.5				
									(all μg/ m^3)	62-99		NAS	1974 Air qlty & Auto EmsnCn V2,p250	Strngth of assn bet dmgchd lung fnctn & NO_2 xpsre is rltvly wk, owng to lack of cnsstncy of assctn over ti & absnce of clear dose-rspnse rltn.
									soilindx (Coh's/ 1Mlnlft)	0.86-2.09				
Shy et al	1973 ibid. 1974 EPA/ 650	Cncnnti O-2 areas	394	7-8	M/F	Wh Bl	Uppr Lo'r Mid Lo'r Mid	$FEV_{0.75}$ obtnd 12X fm ea ch, once wkly in mrz Nov. 67, Feb & May 68	TP	61-85 & 96-133	Wh ch xpsd to aver SS lvls had lo'r $FEV_{0.75}$ than wh ch xpsd to lo'r lvls. Bl ch had cnsstntly lo'r rdngs than wh; rdngs did not vary & pllt n xpsre. Prfrmnce in pltd nbrhds imprvd dur assns of lo p but not to lvl of entrpts in lo xpsre nbrhd. Rslts sprt hypothss that chrnc xpsre to rltvly lo lvls SP was assctd c sgnfcntly lo'r vntltry prfrmnce.	NAS	1975 ibid., p. 73	Cites crtcsm by Higgins & Ferris (1974) who point out inconsstncs in data & rltvly sm obsrvd dfrnces, & assrt that "random scatter ans to be a sch more likely xplnatn than air pllt n." [This same crtcsm is lvld at NYC stdy below.]
									SS	7.7-9.1 & 8.9-10.1				
									SO_2 (all μg/ cm^3)	37-50 & 39-57				

APPENDIX A - Continued

Author	Publication	Study Area	Study Population: No.	Age	Sex	Race	Soc-Ec.	Study Method	Pllt Mars.	Exposure Range	Findings	Critic	Publication	View Expressed
Shy et al	1973 Ibid. 1974 EPA/650	NYC 3 areas	2,364	5-13	M/F	Wh	Uppr Mid	4 rounds of plmnry fnctn tstng (FEV 0.75) cndctd in Nov-Dec 1970, & Jan, Feb-Mar, & Apr 1971 ∈ ch dvdd into 2 age clssfctns bec of hi xpsre dur prev yrs for older ch in 1 clss Rvrhd, LI) nbrhd & 2 intrmed (Queens & Bronx)	TP SS SO_2 NO_2 (all $\mu g/cm^3$)	33-73 10-13 29-71 26-75	ch aged 9-13 xpsd to hi SO_2 lvls & prtclts dur first decde of life had l'r FEV 0.75 than those not xpsd: dfrnce ststclly sgnfcnt for males, but not for fmls. For yngr ch no cnsstnt vartn ∈ pllt xpsre. Dcrmnts in fnctn modst, rnging fm 2-3.4%. Blvs that fact that imprmnt tk place at early age, prior to more severe insults sch as cig smkng & occptnl xpsrs, rndrs fndngs "all the more sgnfcnt in trms of dis prvntn." [this obsrvtn applies to fndngs frm stds in 3 cities.]	Wehrle & Hammer	1974 AMA Conf	Very favrbly impressd ∈ stnds of Shy in 3 Am cities, sttng that "they hv clrly dfnd a cnsstnt rltnshp bet imprd vntltry fnctn & xpsre to incrsd lvls of SO_2 & prtclts." Also that early xpsre to elvtd lvls for prds of 5 yrs was accm nd by prolngd dfency of vntltry fnctn.
												Rall	1974 EPH 8: 117	Unfvrble crtcsm based mainly on prsntn of insffcnt dtls. "All in all, it is not at all clear
												NAS	1974 Ibid. V2, p457	that the dfrnces in lng fnctn bet nbrhds wh were shown in these stds can be attrbtd to d rnces in air pllt bet them."
												NAS	1975 Ibid. p. 72	States that maj dfclty ∈ stdy is eqpmnt used, tho lg nos prhps overcm this. Fndngs rqure rpletn, prtclrly in vw of gr imprtnce of dtrmng whthr costly cntrl msrs hv led to a dcrse in advrse h effcts of air pllt.
												NAS	1973 Conf. H. Effcts Air Plltnts p. 424	The argmnts cncrng the med imprtnce of a slight albeit ststclly sngfcnt decrease in crtn rltvly crude msrmnts of vntltry fnctn seems to me valid.

NOTE: Full references for published reports given in Literature Cited.

Compacted spelling necessitated by space limitations.

APPENDIX B-1

REPORTS OF PREVALENCE OF UNDUE ABSORPTION
OF LEAD AMONG AMERICAN CHILDREN
(Partial List)

Investigator	Pub. Year	Study Population	Study Method	Findings Reported
Anderson & Clark	1974 EHP 7:3-6	2,580 ch aged 106 living in dilpdated or progressively deteriorating housing in 31 cmnts rur & urb (DHEW study)	Screening program to detect extent of lead poisoning in presence of exposed lead-painted surfaces; blood samples analyzed by anodic stripping voltametry method; homes of those c elevated blood lead levels screened c portable x-ray fluoerscence analyzer; retest & confirmatory blood lead analysis for ch c concentration eql to 40 ug/100 ml; & 35 ug for ch < 3	In 25 communities c 2,269 ch. approx. 5% had confirmed blood lead elevations (6 communities & 311 ch excluded from report because of missing data). Approx. equal nos. of black & non-black ch were screened, ab. 38% were < 3 yrs. Rates for younger ch were somewhat higher (6.0 vs. 4.8%) in both groups, while rates for black children were markedly higher in both age groups (7.6 vs. 3.0%).
Balto. City H. Dept.	Lin-Fu 1973	No. ch not specified	Screening 1966-1970	43.3% of ch c bl ld lvls of ≥60 ug/100 ml came from outside so-called inner city.

APPENDIX B-1 - Continued

Investigator	Pub. Year	Study Population	Study Method	Findings Reported
Caprio et al	1974 Arch EnvH 28:195-197	5,226 ch aged 1-5 in Newark, randomly distributed c no age bias evident	Analysis of blood lead made by atomic absorption spectrophotometry & plotted on base map according to (1) residential distance fm. roadway, & (2) residential exposure to aver. weekday vehicle densities	Of entire sample, 35% had blood lead levels considered actionable (bet. 40 & 59 ug/ 100 ml). The nearer the residence to maj.. arteries or to vehicular density, the greater the risk of actionable blood lead concentrations (an increase of 66-77%). "The danger of this particular env. hazard is particularly shocking when one analyzes the differences in rates (2x) of excess lead absorption (at 60 ug/100 ml.) as a function of traffic densities (aver. weekday vehicular densities exceeding 24,000 vehicles)."
Cohen et al	1973 JAMA 226: 1430-1433	502 ch mean age 4 yrs. 230 ch in rrl Dutchess Co., N.Y. & Litchfld. Co., Ct. & 272 urb ch in Hartford, Ct.	Env., socec & hstrcl data; also blood lead levels & hematocrit rdngs c repeat bl ld dtrmntns prfrmd on all ch c initial ld lvls of ≥40 ug/ml; houses & play areas of all rrl ch c ld lvls of ≥40 ug mnd; intrr paint samples for urb ch & tap water for both groups xmnd	Mean bl ld lvls of rr group markedly lower than that of urb group (22.8 vs. 32.7 ug/100 ml). 9% of rur ch had lab evdnce of ≥40 ug/100 ml comprd to 23% urb. "Prev. urb stds hv emphasized role of race, ethnic group, socec status, hsng, famly size, prntl seprtn, hist. pica for paint as cse of incrsd ld intake: most of these factors except for pica were not found to be signfcntly rltd to bl ld lvls in rur ch."

178

APPENDIX B-1 - Continued

Investigator	Pub. Year	Study Population	Study Method	Findings Reported
Fine et al	1972 JAMA 221: 1475-1479	6,151 ch aged 1-6 in 14 Illinois cities of intermed. size (10-150,000 pop.); mean of 250 ch/city	Blood specimens xmnd with atomic absrptn spectrophotometer dur. summer 1971 fm. cities slctd on basis of xprssd desire to prtcpte & on estmtn of ablty to cmplte prgrm sccsflly	Pdtrc ld intxctn is not confined to urb slums, but does occur c alarming frqncy in smaller cmnts. Of totl tested, 1,147 (18.6%)--rprsntng 6.14% of totl pop 1-6 yrs. of age--were fd to hv evdnce of undue absrptn of ld (>40 ug/100 ml). Prcntge varied from 9.6-31.3% in ea city, c the mean bl ld lvls of samples fm dfrnt dities rngng fm 19.8-32.9 ug/100 ml. Nearly one half of ch dmnstrtd bl ld vals in range of 20-39 ug, while only slightly more than a third showed vals. <than 20 ug/100 ml.
Lin-Fu	1972 NEJM 286: 702-710	213,988 ch high-risk areas in 7 mtrpltn cntrs aged 1-6 yrs.	Review of rslts of screening prgrms in old nbrhds, c bl ld dtrmntns prfrmd in late 1960s & 1970-1971; evdnce of undue absrptn based in main on single finding	Bl ld lvls of ≥40 ug/100 g found in from a low of 20% of ch (in Chicago) to a high of 38.9% (in Newark). Bl ld lvls of >60 ug/100 g found in from a low of 5.9% (in NYC) to a high of 14.9% (in Phila). ". . . in magnitude, the problem of undue absrptn of ld amng ch lvng in old nbrhds is matched by few, if any, oth ped public health prblms."
	1975 Toronto Conf. pp. 8-11	1,002,360 ch aged 1-6 yrs in 8 DHEW rgns	Nationwide screening under Chldhd Ld Poisoning Cntrl Prgrm Fiscal yrs. 73, 74, 75	6.42% had confirmed bl ld lvls of ≥40 ug/ml, but in FY 73 24% (more than 8,000 ch) did not return for cnfrmty test, & in FY 74 14% (4,200 ch) did not return.
		277,346 ch aged 106 yrs in 8 DHEW rgns	Nationwide screening (same as abv) FY 73	7.17% had confrmd bl ld lvls of ≥40 ug/ml (minimum pstv findings in some areas but higher in others: i.e., 30.8% of 5,731 ch in Milwaukee & 19% of 2,885 ch in Erie Co.,NY)

APPENDIX B-1 - Continued

Investigator	Pub. Year	Study Population	Study Method	Findings Reported
		58 cmnts in dfrnt geog. areas rngng fm 5-100,000 pop.	Limited surveys by DHEW (Anderson & Clark and Simpson et al) bet. 1971 & 1973	Udue absrptn of ld among ch is a geographically widespread phenomenon fd in smaller communities as well as lg urb cntrs; in rural area, & in middle & upper middle-class as well as ghetto homes.
Natl. Bur Stndrds	1972 Pub. TN-746 cited by Lin-Fu 1973 NEJM 289: 1230	344,657 ch	21 screening programs bet. 1969-1971	8.8-32.9% had bl ld lvls of ≥40 ug/100 ml (overall aver. 26.1%).
Needleman et al	1972 Nat. 235: 111-112	109 asymptmtc ch in Phila (69 ghetto, 49 sbrbn) aged 6-7	Xmntn of ld lvls in dcds teeth dtrmnd by atomic absrptn spectrophotometry	Residents of lead belt had 5x mean cnctrtn of whole tooth ld comprd to sbrbn cntrpts (mean tooth ld for sbrbn ch 11.1± 14.8 ppm; for ghetto ch 51.1± 109.0 ppm). Amng sbrbn ch, 11 had tooth ld lvls of 2 ppm or less, & 4 had lvls below 2 ppm, & 1 ch had lvl of 110 ppm. "These findings may be due to dfrncs in atmsphrc ld in the city & sbrbs. In areas where ld eating by ch is frqnt, tooth ld lvls are elvtd."

APPENDIX B-1 - Continued

Investigator	Pub. Year	Study Population	Study Method	Findings Reported
Needleman & Shapiro	1974 EHP 7:27-31; NEJM 290: 245-248	760 asymptmtc 1st-graders fm 2 schl dstrcts in Phila (1 consdrd high risk, oths. low risk for ld xpsre)	Xmntn of shed dcds teeth for ld by anodal stripping voltammetry plus analysis of samples of dust fm inside schls (totl 14), playground & gutter dirt	"These data portray a prvlnce of xpsure to ld in at-risk groups greater than heretofore rptd. They also show that oth groups than those ordnrly acknwldgd hv elvtd bdy ld burdens." In low-risk area, 3% of pblc & 6.6% of prochl stdnts had dentine ld cnctrtns greater than 100 ug/g contrstd c 66% of bl pblc schl stdnts (lvng in older dtrrtd homes) & 43% of white prochl stdnts in dirtier area (houses old but in gd repair). Dust & dirt cncntrtns (fm indstrl & autmtv srcs) "substantially" grr in high-risk area.
Phila. Dept. Pub. Health	Lin-Fu, 1973	No. ch not specified	"Recent" report	Approx. 50% of rptd cases of chldhd ld poisng located outsi of inner city; sm fm census tracts where prprty vals were equl to or grr than aver prprty vals of most dwelling units in City of Phila.
Rueschel et al	1972 JAMA 222: 462-466	705 ch in impvrshd sctn of Boston aged 6 mos - 6 yrs	Ld in hair (fm 20 mg sample) tkn in house-to-house survey analyzed by atomic absrptn spctrscpy, fold. by dtrmntn of venous bl in suspicious cases	98 of 705 fd to hv incrsd ld burden (excdng 0.5 ug/gm). Mean ld hair cnctrtn of 118 (in cntrst to 27 ug/gm amng ch in Grr. Boston area), c 44% xhbtng vals of 100 ug/gm & greater. In 343 ch whose bl ld was also msrd, mean ld cncntrtn was 0.37 ug/gm. Early rcgntn of such ch is "paramount, since failure to identify them can be severe."

APPENDIX B-1 - Continued

Investigator	Pub. Year	Study Population	Study Method	Findings Reported
Simpson et al	1973 HSrv Rpts 88: 419	50-150 ch aged 106 in ea of 27 cities (DHEW study)	Xplrtry screening prog. amng ch lvng in older hss where it was likely that ld paint was on intrr walls. Homes screened c prtble x-ray fluorescence analyzer	Ch c elvtd bl ld lvls fnd in 25 cities. Hazardous ld paint (>2 mg/cm^2 on dwelling surfaces) accessible to ch thruout nabrhds fm wh ch were screened.
Dept. H, Ed, & Welfare (DHEW)	1972 Pub.73-10002 cited by Lin-Fu 1973	2,309 ch	Survey in 27 cities in 1971-1972 in midwest, south, west, north & east	Ch c bl ld lvls of ≥40 ug/100 ml found in all but 4 cities (in cities where no sch ch were fd, only a very lmtd no. were tested).

NOTE: Full references for published reports given in Literature Cited. Compacted spellings necessitated by space limitations.

APPENDIX E-2

ANIMAL STUDIES ON EFFECTS OF LEAD EXPOSURE ON YOUNG
(Partial List)

Investigator	Pub.	Study Population	Study Method	Findings Reported	Critic	Publication	Views Expressed
Allen et al	1974 ENP 7:239-246	Infant rhesus monkeys cmprd c adlscnt & adlt monkeys	Xpsre to ld prvld by addtn of ld acetate (0.59 mg/kg) to frmla or by cnsmptn of ld prtcls fm ld-based srgte mthrs	"These data sgst that, like man, the infnt nonhan primate is much more sscptble to ld intxctn than is the adlt." Smptms of ld intxctn dvlpd wthn 6 wks. in infnt mnkys. C ld lvls bet 300-500 ug/100 ml, seizures, mscle trmrs, & altrd socl interaction were prdmnnt chgs, C vsl imprmnt in more svrly affctd. In anmls C bl ld lvls <100 ug, hyperactvty & insmnia were obsrvd. C elmntn of xpsre, seizrs sbsided & vsl imprmnt was rdcd, but abnrml socl interactn prsstd. Smlr xpsre to adlscnt & adlt mnkys prdcd ld lvls in xcss of 200 ug but no obvs bhvrl abnrmlts.	EPA	1973 Postn Paper IV-7	Though low dosgs admnstrd to the mnkys were vell in excess of those found in human ld poisgn, the dpndnce of respnse upon age is noteworthy.
Carson et al	1974 ENP 7:233-237	Mature sheep & lambs prenatlly xpsd to ld	Bhvrl txclgc tchnqs emplo.ed to assess effcts of ld in var. doses on CNS in mat. sheep, & on lambs xpsd to var doses dur gsttn & postnatlly	"Slowed lrng was dmnstrtd in lambs prenatlly xpsd to mtrnl bl ld lvls of 34 ug/100 ml dur gsttn wh n tstd on nonspatial, two-choice vsl d.. mntn prblms at 10-15 mos. of age.... The rslts of our xprmnt are cnsstnt C clncl rpts of the nrlgc effcts of ld poisnng in ch.... These rslts hv dmnstrtd vsl lrng dfcts in lambs C bl ld lvls blo the 40 ug/100 ml rcmndd by EPA" in 1972.	EPA	Ibid. IV-6	This deficit is consstnt C vsl-preptl prblms that hv bn noted in ch C ld poisng. Xtrpltn of these findings drctly to man is, however, smwht uncrtn since a bl lvl of 34 ug/100 ml in sheep may be eqvlnt to a hi'r bl ld in man.
Grant & Howard	1975 ENP 10:267	Rat pups fm 1-30 days of age, 2-3 pups of litter srvng as cntrls & 5-7 rcvng ld in milk soltn	Ld acetate in var. doses admnstrd via stmch tube & tstd at 40-45 & 80-90 days of age for ld effcts on food & water cnsmptn & bdy wt gain, genrl locomotor actvty, aggrssvnss twd mice, & lrng in apttvly-motvtd lrng tasks	Most sallent ftrs: (1) dmnstrtn of sxl dchtmy in rgrd to ld-indcd hyperactvty, C males dsplyng syndromo mch more mkdly than fmls--anlgs to prvlnce df hyperknte syndromo among male hum ch; (2) dose-dpndnt rltnshp bet hyperactvty & acquisi ld trtmnt; & (3) prsstng nat of hyperactvty into adlthd lng after csstn of xpsre to xgns ld.			
Kostial et al	1971 Nat. 233: 564	29 rats 5-7 days old	Artfcl feedng for 8 hrs. C cow's milk plus $CaCl_2$ & KH_2PO_4 & carrier-free ld-203 & then rtrnd to mthrs; 12 killed at 40 hrs, 17 at 80 hrs after start of xprmnt, & radioactvty dtrmnd in whole bdy & after rmvl of g-i tract	"The prncpl finding ... rmns the very hi absrptn of ld fm the intstne of newborn rats... If we assme that the % of intstnl ld absrptn is not inflncd by dose, we cld xpct mch more ld to be absrbd fm the intstne in the yng than genrlly spsd. If these vals are xprssd per kg bdy wt, the dfrnce in the amt of ld absrbd per unit wt in yng & adlts wld be even grr.... Our rslts cld also be impt for dtrmng criteria for hum safety fm cntmntn of the ambient			

Investigator	Pub.	Study Population	Study Method	Findings Reported	Critic	Publication	Views Expressed
Cont. Kostial et al				atmsphere c̄ ld, wh might hv to be cnsdrd for yng ch when cmprd c̄ adlt vals."			
Michaelson & Sauerhoff	1974 EHP 7:201-225	Sletd xprncd breeder albino rats (Forter strain) for sur frms ld encphlpthy & timed orgnt Sprague-Dawley rats for less svr & yng of both groups c̄ cntrls.	c̄ ld acetate in dfrnt amts added to chow of mthrs immed upon b of yng, msrd mthrs fd cnsmptn & bdy wt chgs & bl, milk & brain ld cntnt; also newborns bly & brain wt chgs, bl & brain ld cntnt & var brain nrochmcl cmpnts as well as spntns actvty	"Ld-poisnd newborns hv prnncd rtrdtn of growth & dur the 4th wk of life dvlp the svr signs of ld encphlpthy..." This occrs after gainng access to ld-cntng diet. When mthrs diet is chngd at day 16 to lo lvl ld, sckings still hv rtrdd growth but do not dvlp prplgia or grossly apprnt crbllr dmge; however, dur. 4th wk, they xhbt less svr form of encphlpthy, cnsstng of hyperactvty, aggrssvnss, tremors, & preoccptn c self-grooming. "It is possble that our findings on incrsd motor actvty & chgs in brain dopomine may crrspnd to early rspnss of ld xpsre bef the usual lkd for overt signs of ld toxcty."			
Millar	1970 Lancet 2:695-698	Female rats c̄ cntrls & suckling rats	Adlt rats fed on stndrd diet c̄ 40% ld acetate added, cntrls c̄ no ld added; yng rats serfed at 30-40 days. & tissue ld msrd in var orgns	An 80-90% rdctn was obsrvd in actvty of ALA-dehydratase in both bl & brain, & a 60% rdctn in liver, & a sgnfcnt crrltn in suckling rats bet bl & brain ALA-dehydratase actvty. "These rslts sggst that even modest elvtans of bl-ld may be assctd c̄ biochmcl abnrmlts in the ch brain."			
Nathanson & Bloom	1975 Nat. 255: 419-420	Adlt male Sprague-Hawley rats	Stdy of enzyme actvty in cerebllr homogenates under var cncntrtns of ld; in nrml diet (0.5-1 μM), at 3 μM, & at 100 μM	"... very low cncntrtns of ld inhbt adenyl cyclase actvty in main brain tissue ... the mrkd inhbtry effct of lo cncntrtns of ld on an enzyme thought to be intmtly invlvd in synaptic transmssn sggsts that intrfrnce c̄ cyclic AMP mtblsm by this ion cld be a factor in sm of the nrlgcl mnfattns of ld toxcty."	Needleman	1975 Testimony Cal. Air Rsrcs Board Sacramento	"This stdy often locates a dstrbnce in actvty of a crtcl brain enzyme at xtrmly sm cncntrtns of ld lvls fnd in 'nrml" hmn popltns."
Silbergeld & Goldberg	1974 EHP 7:227-232	Adlt female mice c̄ cntrls & suckling mice--totl over 150 anmls fm over 30 litters at 4 dfrnt tis dur c'ndy yr	Ld acetate gvn as drnkng w to test group, sod acetate to cntrls (suckling thru xpsl thru mthrs milk & at weaning thru w); growth & dvlpmnt mtrl & lvls of motor actvty msrd; drugs crntly used in trtng hyperactvty in ch also evltd	Growth & dvlpmnt "sgnfcntly" affctd in offspring of mthrs gvn ld soltns to drink. Ld-trtd mice were more than 3X as actv as age-mtchd or size-mtchd cntrls. CNS stmlnts sprssd hyperactvty in ld-trtd mice, while phnbrbtl xrcbtd it (cntrl anmls rspndd as xpctd). "Ld-indcd hyperactvty was not apprntly dose-rltd, at lst not in range of doses used ... This wld sggst that incrss in motor actvty may be an early smptm of ld poisng assctd c̄ lo lvl intxctn."	Needleman	Ibid.	"Most prtnnt to the cncrns of pdtrcns, nrlgsts, & edctrs is the finding by Silbergeld that the rspnse of these mice to phrmclgc agnts sch as amphtmne, rtln, & phnbrbtl xctly prlld that fnd in hyperactv ch."

NOTE: Full references for published reports given in Literature Cited.

Compacted spelling necessitated by space limitations.

APPENDIX B-3

STUDIES ON EFFECTS OF LOW LEVEL LEAD IN CHILDREN
(Partial List)

Investigator	Pub. Year	Study Population	Study Method	Findings Reported	Critic	Publication	Views Expressed
Albert et al	1974 EHP 7:33-39	371 mostly bl ghetto ch aged 5-15 yrs. (mn9) 211 c̄ no diag. poisng. & no therapy. 160 c̄ hosptlzd poisng & thrpy	Sample dvdd into 5 xpsre catgrs (bl & tooth lvls) & cmprd on basis of valdtd illnss xprnce, psychmtrc tests (for 159), & acdmc prfrmnce	Xcpt for diagnsd poisng cases, elvtd bl ld (≥0.06 mg-%) excss ld xpsre (hi lvls tooth ld), no dltrs h effcts. Invstgtrs blv findings raise 2 pts: (1) doubt as to sgnfcnce to h within first 10 yrs life of rising atmsphrc ld lvls (tho nmrs env factrs in ghetto may mask effcts(, & (2) possble value of chltn trtmnt for all elvtd bl lds c̄ or wtht symptms.			
Baloh et al	1975 Arch. Neur. 32: 326-330	54 ch over 3.5 yrs 85% bl, class V, 27 asymptmtc but c confrmd ld absrptn (50 ug/100 ml) 27 cntrl ch (30 ug/ml) matchd for age in mos, sex, race, socec status	Evltn of ea ch incldd: dplete bl ld dtrmntns, cc̄p hist, phys. xm, qnttv nrlgcl tsts, & cmprhnsv psych tests (portions of WPSSI or WISC tests, etc).	Mn IQ of 2 groups smlr. Only sgnfcnt dfrnce bet groups concrnd overactv bhvr--44% of elvtd group, 15% of cntrls (mthrs of more ch in elvtd group rprtd overactv bhvr, but thr nrlgst nor psychlgst in objc's bhvrl ratings found sgnfcnt dfrnce). Elvtd group tndd to do poorer on tests rqrng fine motor ablty, but not sgnfcnt dfrnce in psych tests. ("Possible" crtcsm by invstgrs: lack of demnttn of ld absrptn in infancy in both groups.)			
Beattie et al	1975 Lancet 1:589-592	154 ch age 2-6 yrs. 77 mntlly rtrdd 77 non-rtrdd cntrls closely matched for age, sex, socl class, & type of infnt feeding	Bl ld dtrmntns & tap-water ld msrmnts in pres. homes & in homes mthrs occpd dur. prgnncy & ch occpd dur. yr 1	Water ld contnt "sgnfcntly" higher in rtrdd group, & prbblty of mntl rtrdtn "sgnfcntly" incrsd when water ld lvl excdd 800 ug/l. Bl ld lvls also "sgnfcntly" higher in rtrdd group. "Ld cntmntn of water may be one factor in mltfctrl etlgy of mntl rtrdtn . . . "	Needleman	1975 Testimony bef. Calif. Air Resources Board Sacramento	"In a rcnt rpt, Beattie, et al, idntfd ch in Glasgow c̄ idiopathic mntl rtrdtn. These were matched c̄ nrml ch on age, socec status & ggrphcl rsdnce. The water sply of the home of the mthr, while she was prgnnt c̄ the indx case, was vstd & the water anlyzd for ld. No nrml ch cm fm homes c̄ xcssv ld in their water. The authors cnclde that lvng in a home c̄ hi water ld incrss the risk for rtrdtn by a factor of at least seven."
de la Burde & Choate	1972 JPed 81: 1088-1091	142 ch age 4 yrs. 70 c̄ xpsre to ld 72 c̄ cmprbl socec backgrnd but "prsmbly wtht xpsre"	Series of psych tests in 5 areas: IQ fine motor dvlpmnt, gross motor dvlpmnt, cncpt frmtn, bhvr; also urinary coproporphyrin tests in both groups, & in addtn in xpsd group bl ld dtrmntns & radiographs of lng bones & intstns	Mjrty ch in ea group had aver. IQ. 35% ld xpsd (bl ld lvls. 0.04 mg/dl but no clncl symptms) prfrmd nrmlly in all tests, cntrstd c̄ 65% cntrls. Dfcts occrrd most freq. in fine motor fnctn & bhvr. Dytns in bhvr ratings: amost 3X as often in ld xpsd. Invstgtrs' vw: Dfrncs fd may be due to env. factors other than ld, since ld lvls in cntrl group were not dn--slctn based on their prsmbly ld-free envs. sprtd by neg. coproporphyrin urine tests.	Klein et al	1974 AJDCh 127:806	"Unfortunately, the ch in the cntrl group did not hv bl ld dtrmntns & were not known to hv pica. . . . It is possible that pica, rather than ld, is the varble that rsltd in the dfrncs bet the two groups."

APPENDIX B-3 - Continued

Investigator	Pub. Year	Study Population	Study Method	Findings Reported	Critic	Publication	Views Expressed
de la Burde & Choate	1975 JPed 87: 638-642	Same group as abv c 6 xcptns 137 ch aged 7 yrs. 67 c asymptmtc ld xpsre bet. ages 1-3 yrs. 70 cntrls (sa. age, socec backgrnd) & "prsmbly no sgnfcnt xpsre"	Thoro pdtrc nrlgcl evltn at 7 yrs, rptd at 8 yrs for 58 ld-xpsd & 58 cntrls; also series of psych tests, xm schl rcrds, & analyss of dcds teeth for ld cnctrtn	Xpsd ch had dfcts in global IQ & assctv ablts, in vsl & fine motor crrdntn, & in hbvr. Schl fail. due to lrnng & bhvr prblms was more freq in ld-xpsd than in cntrls. "Most sgnfcnt finding": dfrnce in bhvr ratings bet 2 groups, c bhvrl prblms bcmng "obvious & dstrbng" in schl setting.	Needleman	1975 Testimony bef. Calif. Air resources Board Sacramento	"Dr. Brigette Burde has shown that ch c known pica, but no symptms when studied later in life hv a hi'r incdnce of bhvr dsrdrs & prcptl dfct."
David et al	1972 Lancet 2:900-903 1974 EHP 7:17-25	119 ch aged 3.5-12 yrs. 82 dvdd into 4 groups ("pure" hyperactv, hi'ly prbbl cse for hyperactvty, possble cse, ld poisng) 37 cntrls	Cmprsn of body ld by 2 msrs: bl ld lvls & urine lvls after challenge by single oral dose of penicillamine. Hyperactvty msrd 3 ways: MD diag, tchr's rating, & parent questionnaire	Hyperactv ch had "sgnfcntly" hier vals. in both msrs. than did cntrls. More than half hyperactv ch had bl ld lvls in raised (but not "toxic") range, & 60% of post-penicillamine urine lvls were in "toxic" range. ". . . argument for considering any ld elvtn above 24.5 ug/100 ml should receive serious attention."	Baloh et al	1975 ArchNeurol 32: 329	"As in mntlly rtrdd ch, however, ld absrptn cld be scndry to hyperactvty rather than the cause."
					Klein	1974 AJDCh 127:806	". . . absnce of pica as a controlld varble prvntd dsgntn of a cmusal rltnship."
					Malcolm	1974 ProcroySocMed 67:168	Crtcsm idntfctn of hyperactvty by bl ld lvls bec of "analytcl & cntmntn dfclts, & the assmnt of hyperactblty is to a lg xtnt sbjctv. I do not at prsnt find this rpt very cnvncng, c a fairly sm tho statclly sgnfcnt dfrnce in the mn bl ld lvls c rltvly lg stndrd dvtns."
					Needleman	1975 Testimony,Cal.	"This stdy shld be cmprd to that of Drs. Silbergeld, Michaelson & Sauerhoff who prdcd hyperactvty in the immature rodent c ld."
Feldman et al	1973 AmJ Dis Ch 125: 39-41	46 ch c known plmbsm (idntfd by 1 or more of 3 criteria) & 22 cntrls aged 19 mos-10 yrs c no clncl or lab evdnce of incrsd ld brdn	Motor nerve cndctn vlcty (incl. latencies & mscle action ptntls) detrmnd by applctn of mthd of Hodes et al	Ch c plmbsm were fd to hv rdcd mn motor nerve cndctn vlcts as cmprd c nrml ch. Mild unstdnss of gait, frqnt falling, or rdcd dsre to run or play may be only symptms of neuropathy. "This finding may prvde a vallie test for dscvrng mnml prphrl neurotxc effects of chrnc xpsre to ld ingstn."			
Kotok	1972 JPed 80: 57-61	49 ch aged 1-5.9 yrs. 24 ch c elvtd bl ld lvls (0.058-0.137 mg%) 25 cntrls (0.020-0.055) fm smlr inner-city env cmprd c sample fm hi'er socec lvl used in Dnvr Dvlpmntl Screen. Test	All ch dvdd into 3 groups accrdng to neontl cndtn & phys/emtnl env, based on hist., obsrvtn of ch c parents, & med rcrds; then DDST gvn to all ld & cntrls, & cmprd c aver prfrmncs of Dnvr ch	Ch in both ld & cntrl--in cmprsn c Dnvr--group dmnstrtd diffs in fine motor-adptv & lang fnctns, but not in gross motor fnctn. No sgnfcnt diffs in dvlpmntl scores bet ld & cntrls. "Diffs drctly rltd to inadqcs in the ch's env," not to ld toxcty, c a "drct crlltn bet the deg. of env dsrptn & the deg of dvlpmntl delay."	Lin-Fu	Personal communication 2/24/76	Controls poorly chosen.
					Needleman	1973 SmnrsPsych 5:49	"Aside fm the sm sample size & coarse grained dvlpmntl tstng instrmnt (Denver Dvlpmntl Screening Test), the cnclsn of the stdy shld be vwd c skptcsm, since the mn bl ld lvl of the sbjcts tkn as cntrls was 38 ug/100 ml."
					Waldron & Stöfen	1974 Sub-Clinical Ld Poisng, Acdmc Press, 127	Valdty of cnclsn has bn qstnd. Sgst that "it wold hv bn more stsfctry had totl bdy brdn bn estmtd by chltn stds. It is also unstsfctry to cmpre the dvlpmnt of this group of lo'r class, mainly coloured ch c the mainly wh, mid-class ch used in the Denver study."

APPENDIX B-3 - Continued

Investigator	Pub. Year	Study Population	Study Method	Findings Reported	Critic	Publication	Views Expressed
Landrigan et al	1975 Lancet 1: 708-712	124 ch age 3-15 yrs. 46 symptm-free & bl ld 40-69 ug/100 ml (mn 48) 78 ethnc & socec smlr cntrls, & lvls <40 (mn 27): all lvng wthn 6.6 km ld-emttng smltr	Battery of nrlgcl tests admnstrd to all ch >5 yrs; psych assmnts md & Wechsler intllgnce scale gvn to 70 >5 yrs. Venous bl sample prfrmd at ti of nrlgcl xm, & anlyzd in dup by atmc absrptn spctrphtmtry; also med hstry re pica & hyperactvty	"Our findings indcte that chrnc absrptn of prtclte ld sfcnt to prdce bl lvls of 40-80 ug/100 ml may rslt in a sbtle but ststclly sgnfcnt impairment in the non-vrbl cgntv & motor skills ... & sbclncl impairment in the fine motor skills msrd by the finger-wrist tapping test." Testing & WIS for schlch & pre-schlrs showed age-adjstd prfrmnce IQ to be "sgnfctntly" dcrsd in group & higher ld lvls. Ch in all ages in the ld group also had sgnfcnt slowing in finger-wrist tapping test. Full-scale IQ, vrbl IQ, bhvr, & hyperactvty ratings did not differ.	Bryce-Smith & Waldron	1974 Lancet 2:44	"This rpt apprs strngly to reinforce the rsrvtns wh we xprssd cncrng the authors' mthdlgy & cnclsn [rfrnce to wk of Lansdown et al]."
					Lin-Fu	Personal Communication 2/24/76	Quite careful study, but one defect: control children were 1 yr. older (mean age 9.3 vs. 8.3 yrs); did age difference play a part?
					McCabe	1974 Lancet 2:896	Faults stdy on fol grnds: (1) mthd of slctn of stdy pop; (2) size of stdy group; (3) poor mtchng; (4) mthd of intrprtin of dfrnces bet groups; (5) test used for preschlrs.
					Needleman et al	1975 Lancet 1:751	"We cld find no stmnt, rpt, or stdy by an indstry-spnsrd scientist, whthr in-hse grantee, or acdmc consltnt, wh frankly acknwldgs that lo-lvl xpsre is hzrds [in crtcsm of McCabe's crtcsms]."
Lansdown et al	1974 Lancet 1: 538-541	394 ch 119 under age 6, 275 fm 6-16 yrs lvng in area xpsd to "undue" amts of ld	Wechsler stndrd tests of intllgnce & rdng attnmnt (Burt rearrngd) gvn to all schl ch, tchrs rpt of bhvr based on qstnnre of "known rlblty & valdty," & h-vstr info re lngth of rsdnce, f's odcptn, & prsnce of family mmbr who had wrkd at smltng wks dur ch's lifetl; ld dtrmnd in dup in smpls of whole bl by atmc-absrptn spctrophtmtry	"Dstnce fm fctry prdcng ld pllth was rltd to bl-ld lvl, but there was no rltnshp bet bl-ld lvls, gnrl intllgnce, rdng ablty & any msre of mntl fnctng. L'r lvls of intllgnce & hi'r rates of dstrbnce fd to be more rltd to socl factors." No psychlgcl tstng of ch <5 yrs bec "of the unrlblty of psych tstng below schl age."	Bryce-Smith & Waldron	1974 Lancet 1:1166 et seq	Faults stdy on a no. of grounds: (1) bl ld msrmnt lmtd to current, rather than to past, xpsre; (2) no ch stdd; (3) no bhvrl assmnts by psych (only by tchr); (4) no rpt. re progrss s. sch; (5) cnclsn illgcl & mjstfd on basis of data prsntd. Dplre "haste to embrce socl factrs as main cse."
					David	1974 Lancet 1:866	Cnsdrs findngs biased, & crtczs "the emprcl sprt prsntd for the cnclsn that there is no rltnshp bet raised bl-ld lvls & bhvr dstrbnce is srsly open to qstn." Crtczs mthdlgy in svrl areas: (1) bl ld dtrmnts; (2) assmnt bhvrl dstrbnce, (3) age & sex analyss.
					Landrigan et al	1974 Lancet: 1:1167	Dplre lack of adeq cntrls, wh mks it dfclt to assess the valdty of cnclsn rchd.
					Lin-Fu	Personal Communication 2/24/76	2 faults: test not sensitive enough to determine effect of lead, and only single blood ld level taken.
McNeil & Ptnasik	1975 Intrntl Symp Yugoslvia	138 ch age 21 mos.-18 yrs (med. 9 yrs), chrnclly xpsd to cndtns rsltng in incrsd ld absrptn 101 c lb ld lvls 40 mcg%; 2 cntrl groups (1 fm El Paso, oth fm rur cmnty) matched for age, sex, ethnc backgrnd & incme	Eval incldd hist qstnnre, slctd lab stds, genrl phys xm, nrlgcl xm, nerve cndctn tests, psychmtrc evltn, & tchr evltn. Test grp sbdvdd into 7 grps on basis of 3 sets of criteria: loctn, bl ld lvls, & xpsre risk factrs. Addntlly, 5 pllth xpsre parameters idntfd & subgrps set up acrdng to deg: serious, mod, low	"No evdnce of gross or sbtle injry was dtctd by an extnsv battery of tests in the geogrphc grps, the grps seprtd by ld lvls, or in slctd grps consdrd to be of grtst risk. ... No impairment of fine motor or perptl skills was dtctd on physcl, nrlgcl, or psychomtrc xm. Hyperactvty was not apprnt fm hist. qstnnre, physcn estmte, psychlgcl evltn, or tchr evltn."	Lin-Fu	Personal Communication 2/24/76	Extremely poor study & a no. of fallacies and no. of assumptions; matched ch in areas of residence instead of ld. levels.

APPENDIX B-3 - Continued

Investigator	Pub. Year	Study Population	Study Method	Findings Reported	Critic	Publication	Views Expressed
Millar et al	1970 Lancet 2:695-698	57 hosptlzd ch age 1-12 yrs 30 c nrml IQ (>70) & c illns affctng nthr brain fnctn nor haem mtblsm 27 either c IQ lvls <70 or "obvly mntlly dfcnt"	Intllgnce msrd either by clncl evltn of edctnl cpblty or by frml tstng using Stanford-Binet tchnqe. Lab tests incldd packed-cell vol. & haemoglbn by Dacie & Lewis mthd; bl lv msrd by stndrd dithizone mthd c 1 modfctn; ALA-dehydratase actvty by Lichtman & Feldman	Stdy rvld neg corrltn bet bl-ld lvls & ALA-dehydratase actvty in ch. Sgnfcnt decrease in enzyme actvty occrrd at ld lvls consdrd to be in upper range of nrml (20-40 ug/100 ml). "These findings & the possblty that smlr biochmcl chgs are prsnt in the brain also, mphszs the dngrs of xpsre to even very small amts of ld dur chldhd, & sgsts that a downwrd rvsn of acceptble lvls of bl-ld in ch is dsrble."			
Perino & Ernhart	19[illegible] JLrng Dsblts 7:28-30	80 bl ch aged 3-5 yrs 3 mos all asymptmtc & c no known illnss or hist of cndtns assctd c intllctl impairmnt or ld intoxctn 50 c ld lvls bet .01 [illegible].03 mg 30 c ld lvls bet .04 to .07 mg (c no sgnfcnt vrncs bet grps in sex, age, prntl intllgnce, no. ch, order of birth, & b wt.	Ea ch gvn McCarthy Scales of Ch's Ablts, prvdng fol. scores: Gnrl Cgntv, Vrbl, Preptl Prfrmnce, Qntty, Mmry & Motor Ablts. Ea parent (usually mthr) gvn Quick Test (gross intllctl [illegible]), & info obtnd re prntl ed, socec status, ch's b wt & b order, & no siblngs	"...as ld lvl incrsd, genrl cgntv, vrbl, & preptl ablts dcrsd. Ld lvls were not rltd sgnfcntly to prntl intllgnce, b order, b wt, & no of sblngs. Ld lvl was, however, rltd to the ed lvl--but not to socec status of parents." Corrltn of parent & ch intllgnce was sgnfcntly dfrnt for ch c ld lvls bet 0.4 to 0.7 mg, but not for ch c ld lvls <.03 mg. "It seems that the criteria set for ld poisoning need re-xmntn."	Needleman	1975 Testimony, Calif.	"In this stdy the crrltn bet intllgnce of mthrs & ch in the lo ld group was .52, a figre smlr to that fd in oth pop stds. The crrltn bet prntl & offsprng intllgnce in the hi ld group, however, was .10, sgnfcntly dfrnt, indctng that anoth factor was oprtv in the dtrmntn of the ch's intllgnce."
Pueschel et al	1972 JAMA 222: 462-466	58 impvrsh preschl ch c "increasd ld brdn" (bl ld >0.5 ug/gm or urinary ld output of 500 ug/24 hrs or both) trtd by chltn therapy & env imprvmnts	Initial cmprhnsv stds incldd ped xm, nrlgcl cnsltn, anthropomtrc msrmnts, evltn of motor fnctn, psych assmnt, nursing & ntrtn intrvws, intnsv ed prgrm, & prtnt lab stds. Follow up 1½ yrs later, reasm focsd on env aspcts & ch's nrlgcl & psych fnctng	Hist. of "mild" C[illegible] symptms (irrtblty & clumsiness) [illegible]d in one-third of ch. Nrlgcl dysfnctn & motor dfcts obsrvd in 22-27%. Intl. psych assmnt rvld lo aver. mntl ablts in maj. of ch; 1½ yrs later a "signfcnt increse" in crtn areas of intllctl fnctng was noted, but not in nrlgcl & motor fnctn assmnts. "The trtmnt c chltng agnts or crctv msrmnts in the env or both, might be in pt rspnsble for the noted gains in IQ points."			

APPENDIX B-3 - Continued

Investigator	Pub. Year	Study Population	Study Method	Findings Reported	Critic	Publication	Views Expressed
Roberts et al	1974 Sci. 186: 1120-1122	1,711 ch & adlts ch aged 0-14 yrs lvng nr scndry smeltrs 286 close to smelter A 1,425 close to smelter B 1,231 cntrls in urb area, smlr socec status	Bl ld dtrmntns (by anodic stripping voltammetry) of totl popltn, & dtld survey of 16 famls (out of possble 30) lvng in hss wthn 150 m of smelter A & B urb contrl famls lvng in hss of smlr age & upkeep	Bl ld cncntrtns of ch (but not adlts) incrsd c prxmty to both smelters. "Bet 13 & 30% of the ch lvng in the cntmntd areas had absrbd xcssv amts of ld (>40 ug/100m of bl & more than 100 ug/gm of hair) as cmprd c 1% in cntrl groups....Mtblc chngs were fd in most of 21 ch slctd frm those c xcssv absrptn; 10-15% of this group showed sbtle nrlgcl dysfnctns & minor psychomotor abnrmlts."			
Roels et al	1975 Toronto Intrntl. Conf.	51 ch lvng in ld smelter area (28 m, 23 f) aged 10-15 yrs 92 cntrl lvng in rur area (28 m, 64 f) mn pd actl rsdnce in both areas 10 yrs.	Stdy of var indices of heme biosynthtc pathway to mod ld xpsre (15-45 ug/100 ml bl) in groups of adlt m & nonprgnt f ld wkrs in addtn to schlage ch. Bl ld msrd by flameless atmc absrptn, FEP by fluorimtrc micromthd, ALAD by stndrd Eur. mthd, & ALA-U by autmtd mthd. Hmtcrt val & hmglbn also msrd	"Sgnfcnt dfrncs xst bet the slope for men & that for wmn or ch; those for ch & wmn dfr not sgnfcntly, but FEP incrse starts in ch at a lo'r Pb-B than in men or wmn...This stdy indcts strngly that ch & wmn are the two crtcl groups to be cnsdrd when env stndrds for ld are prpsd &, moreover, that if incrsd FEP rspnse mst be rgrdd as an unacceptble adverse h effct, Pb-B in ch should not xcd 25 ug/100 ml bl."			

NOTE: Full references for published reports given in Literature Cited.

Compacted spellings necessitated by space limitations.

APPENDIX C

STUDIES ON EFFECTS OF AIR POLLUTION ON GROWTH AND DEVELOPMENT OF CHILDREN

(Partial List of Foreign Studies Published Between 1965-1973)

Investigator	Pub. Year	Composition Study Group	Tests Employed	Pollutant(s)	Findings Reported
Czech					
Antal et al	1968	Matched series of ch*** in 2 towns: hvly pllted & rltvly clean	Unspecified	Lead cncntrtn from 5.2-16.4 ug/m^3 in pllted town to 1.1 ug/m^3 in cntrl town	In pllted town, ch less well dvlpd physclly than mtchd srs in cntrl town. None had clncl ld poisng, but it was assmd that dfrncs seen in growth & dvlpmnt & in intllctl achvmnt owed their origins to hi'r lvl of ld xpsre.
Kapalin	1966	3 groups** Slovak children grades 3-5: group 1 summered in clean recreation area attended boarding school in fall; group 2 lived in highly polluted area but summered in scout camp; group 3 (controlled for social & climatic conditions) lived in less polluted area	Physical exam of body height & structure; lab tests of hemoglobin & red cells	Arsenic	Proper diets & purer air had "levelling effect" both on body structure within each group & on hemoglobin levels, but children from the polluted area continued to exhibit decrease in both hemoglobin values & red blood cell counts.
Kapalin	1967	Children** 7-15 yrs in polluted & cleaner areas	Total of 2,424 Xrays of left wrist & hand (evaluated by method of Gruelich & Pyle)	Not specified	Growth of girls comparable to American girls, but "a delay of about 1 yr. in Czech boys during early school yrs." Demonstrated a "definite relationship between level of growth & osseous age." Children living in polluted areas experienced retarded osseous maturation as compared with those in clean areas.
Kaspar & Tichy	1965	Children** 7-12 yrs	Not specified	SO_2 (up to 1 mg/m^3) dustfall (up to 500-1,000 T/m^2/yr)	Retarded skeletal development is "pronounced" among children in polluted areas; there is also a reduction in volume of individual blood cells & an increase in erythrocyte count. The "incidence of premature fetuses in the region's superficial coal mine basin is statistically significant."
Luhanova	1971	9-10-yr. olds** from polluted & non-polluted areas, ingesting "approximately same food"	Photometric measurement of hemoglobin; cytologic determination of regeneration of mouth epithelium; Whitehead test (separation of amino acids); & determination of protein saturation	Smoke	"All indicators" (the entire physical condition, nutritional condition, & body development) showed that--with "approximately the same diet"--children in dirty neighborhoods were less well nourished.
Petr & Schmidt	1966-1967	7-12 yr. olds** of comparable socio-economic characteristics in high & low exposure communities	Physical exams & lab tests of hematologic indexes (not clinically used in U.S.)	NO_2 & SO_2	Greater frequency of hypertrophied tonsils & cervical lymph nodes & more children w retarded physical development living in polluted areas. Believes that excessive nitrogen oxides combined with sulfur dioxide increase harmful effects in children.

APPENDIX C - Continued

Investigator	Pub. Year	Composition Study Group	Test Employed	Pollutant(s)	Findings Reported
Symon & Petr	1970	Groups*** of children from No. Bohemia living under approximately equal climatic conditions & c comparable nutrition patterns	"Systematic & repeated exams," including blood tests	SO_2, dust, arsenic	2 different types of pollution found "unfavorable" to growth & development of children: (1) significant dust content, lack of untraviolet radiation, but realatively low concentrations of SO_2, and (2) arsenic.
Wokonouva et al	1970	10-yr-olds** living in 3 different areas in Bohemia since birth, categorized by environmental conditions	Lab ests of erythrocytes, homoglobin values, color index, leucocytes, lymphocytes, eosinophils, & serum protein level	SO_2 (0.05-0.5 mg/cm^3) dust (150-500 T/km^2/yr)	Retarded growth & greater morbidity rate for respiratory disease in children living in very polluted neighborhoods.
German Bab et al	1971	321 children*: one group from heavily polluted area; other from nonpolluted distirct	Xrays of left hand correlated c body weight & height	SO_2 & dust (respirable & sedimenting)	"Ossification of the examined bones was retarded in those children growing up in the heavily polluted industrial area."
Dolgner et al	1971	Exposed group of 116 boys & girls aged 9.5-12 yrs; control group of 67 aged 9-12.5 yrs.	Anthropometrical & clinical [illegible], also blood tests	Dustfall	Among those living in more polluted district, a delay in bone maturation & lower erythrocyte count occurred (especially in girls); also, lower hemoglobin values & enlarged cervical & mandibular lymph glands & tonsils; boys showed lower HbE values.
Grosser	1970	Tot. 590,240 ch & 250 cntrls in E. Germany mtchd by age, sex, no sblngs, prsn/rm index, & no appliances in family	30 chrctrstcs incl anthropomtrc data, pulse & bl pressure, erythrocyte & leukocyte counts, alk. serum phosphatase actvty, serum properdin lvl, & vital capacity	Unspecified, but emssn lvls of all rlvnt pllnts must be known & areas must mnfst gr dfrncs in pllnt lvls	Many of 30 characteristics manifest statistically significant changes as a result of effect of pollutants.
Grosser	1971	1st study: 4th & 5th graders**; 2nd study: 1,300 children from grades 1, 5 & 9	Exam of blood, body weight & height, serum phosphatase, electrophoresis, lung volume, & lung function	Unspecified	All measures "more unfavorable" for children from polluted areas.
Nagel	1971	Unspecified	Lab tests	Unspecified	"Air pollution increases the reaction of the lymphatic apparatus & causes a decrease in the red blood cells, thus retarding the physical development of children."

APPENDIX C - Continued

Investigator	Pub. Year	Composition Study Group	Tests Employed	Pollutant(s)	Findings Reported
Pelech	1970	157 boys & 173 girls aged 10-11.5 yrs. in 2 urban & 2 rural areas c & without air pollution	Measurements of body height & weight, bone maturity & respiratory functioning; also lab tests of hematological & biochemical characteristics	SO_2 & dust	Adverse air pollution environemnt considered primary feature of retardation of osseous development, and more significant than influence of different social environment.
Russian Kazasov et al	1971	Children*** near town of Stara-Zagova, Bulgaria	Various laboratory tests	Hydrogen sulfide, nitrogen oxides & ammonia	Pollution produced various biochemical changes: damage to adaption reactions, disturbance of mineral metabolism & oxidation-reduction process, & changes in blood & immunobiological state (including low hemoglobin concentration, etc.).
Makkinya	1972	Children*** in Orsk-Khalilevo metallurgical complex	Adaptometric & electroencephalographic tests	SO_2 & phenols	Pollution produced "significant lag in physical development" in children living in gas-polluted zone, in addition to complaints of headache, dizziness, nausea, epigastric pain, etc.--along c more chronic inflammatory & respiratory ills.
Yelfimova et al	1971	8-11-yr-old Soviet Children**	Unspecified	NO_2 (from 46.3 mg/m^3 to 87 ppm and from 93.5 mg/m^3--176 ppm); also "high" concentrations of sulfur dioxide & hydrogen sulfide & "moderate" concentrations of phenol	"Significant lag in growth, weight, & chest circumference, decreased urinary excretion of vitamin C"--as well as "5-fold excess of upper respiratory infections, 3-fold excess of tonsillitis, & 2.5-fold excess of atrophic rhinitis."
Japanese Hirata	1973	"More than" 250,000 school children*	"Specially designed & computerized" health evaluation method; athletic abilities tests c 50 m run, standing jump, & soft ball throwing	Unspecified	During past 10 years--but especially for period 1970-1972--average height gradually increasing, weight & chest masures gradually decreasing, & jumping capacity gradually deteriorating. Also "excellent healthy specimens & muscular bodies have decreased considerably, & generally frail, unhealthy & fat children have increased in number." High concentrations of smoke & soot in living or school environments produced children with small chest measurements, limited amounts of energy, & poor standing jump ability.
Rokushika & Adyama	1972	25,537 children grades 3-8 in Nagoya City (2 polluted schools, 1 "slightly," 2 clean)	Compared height & physical statistics, & athletic performance c complaints of physical discomfort from air pollution	Unspecified	The complaint rates were higher in polluted schools. "Only significant difference" was noted in running endurance test: children from polluted schools showing "a clearly inferior capacity for endurance." In secondary school children, no relationship between running ability & subjective symptoms, whereas younger children in polluted areas who were under average in physical ability also showed symptoms of bronchitis & neurosis.
Tajima et al	1968	506 boys & girls in grades 5,6,8 & 9 in area polluted by aluminum plant & control only slightly polluted	Xrays of right arm	Fluoride (3-5 ppb) & 100-230 kg/km^2/mo fluorine atmospheric dustfall	5th- & 6th-grade children in polluted area were found "a little behind" in development of carpal bones & in 6th grade in metacarpal & digital phalanx bones; in 8th & 9th-grade girls in polluted area, low levels of fluorine promoted skeletal development. Conclusion: "Pollution in this area does not have serious influence upon the skeletal development of children."

APPENDIX C - Continued

Investigator	Pub. Year	Composition Study Group	Tests Employed	Pollutant(s)	Findings Reported
Tsuji	1973	5th & 6th grade children; also jr. high students**	Xrays of right arm & height & growth rates	Fluorides (atmospheric content 3-5 ppm); in settling particles 100-230 kg/km^2/mo in polluted area & 1/10th these amounts in cleaner area	In polluted area, average height & growth rate slightly greater. Slight delay of maturity in 5th- & 6th-graders. Among junior high pupils, maturity of hand bones slightly faster among children in polluted area. Conclusion: a large quantity of fluoride interferes w/ normal growth of children; small amount seems to enhance it.
Ueki & Tanaka	1971	School children*** in Kuba (cleaner district) & Otaka (highly polluted)	Expiratory flow & ventilatory capacity tests & 2 measures of physical substantiality: Rohrer S & Kaup S indices	SO_2 (mean from 0.38-0.91); dustfall (from 5.8 to 7.35)	Multiplying vital capacity by height in the 3 groups produced no differences, "suggesting that pulmonary function has no effect on level of physical substantiality."

NOTE: Full reference to published reports given in Literature Cited. Compacted spelling necessitated by space limitations.

*Age not specified.

**Number not specified.

***Neither age nor number specified.

APPENDIX D-1

QUESTIONNAIRE USED IN INTERVIEWS WITH ACTIVE PRACTICING PEDIATRICIANS IN "DIRTY AIR" AND "CLEAN AIR" AREAS OF CONNECTICUT

1. As you appreciate, by far the most common type of illness in children--accounting for perhaps 60 percent of acute conditions--is respiratory disease. I presume this is true in your practice?

2. Among the other principal causes of morbidity, what do you see most of in your practice?

A. Infectious & parasitic diseases
B. Gastrointestinal diseases
C. Injuries
D. Chronic impairments
E. Emotional disturbances

(Please rate numerically)

3. In this respect, would you say your practice is typical of that of most pediatricians in the U.S.

If no, how is it different?

4. Do you believe any of the conditions you treat are caused or aggravated by air pollution?

If yes, what ones?

And how and why?

5. Can you recall where you read/heard about this relationship?

A. Pediatric journal
B. Other professional journal
C. Lay press

5. (continued)

D. Conference, professional colleague
E. Other

6. Here is a brief reference to recently published research by an EPA physician. Do you feel that his findings are applicable to your area?

If no, what is your reason?

7. Air pollution has been statistically associated with congenital malformations, as you'll note from this excerpt from the studies of a UPenn scientist. Do you consider this valid?

8. Do you agree with the statement of an EPA physician (Dr. Vaun Newill) that "we're more apt to understate greatly the true impact of pollutant exposure on human health than we are to attribute erroneously many health effects to air pollution"?

9. Here's reference to a recent British study concerning the dangers of smoking in the presence of infants. What's your reaction to this?

10. Do you advise parents to eliminate smoking at home?

11. Do you give any other advice re air pollution from whatever source?

If yes, what advice?

A. Move to cleaner neighborhood
B. Stay indoors when pollution is heavy
C. Wear gas mask
D. Avoid physical exertion
E. Install home filtration system
F. Other

12. Nonimmunized children and battered children are currently featured as urgent pediatric problems. I don't know whether these are problems you face. For example, what preventive health care measures do you recall recommending in thepast couple of months?

A. Safety seats in cars

12. (continued)

B. Improved dietary habits
C. Strict lead-paint housing code enforcement
D. Childproof medicine bottles
E. Cleaner community air
F. Reduced cigarette smoking
G. Safer toys
H. Immunizations
I. Protection for battered children

13. Is much of your time with mothers spent in recommending preventive steps, or do you see yourself as more therapeutically oriented?

Whatever the answer, do you believe other pediatricians are similarly oriented?

14. Getting back to the EPA findings relating air pollution to an increase in respiratory disease, AMA--in this Physican's Guide to Air Pollution--recommends both professional society and individual physician involvement to help upgrade air quality. Is there any way an individual physician can help?

15. I have no extra copies of this Guide with me, but I'd be glad to send you one, if you'd care to have it.

Appendix D-1.1

Exhibit shown during interview

EPA SCIENTIST REPORTS ON RESPIRATORY DISEASES IN CHILDREN

New evidence linking sulfur oxides and suspended particulates to acute lower respiratory diseases in children was reported by a U. S. EPA scientist.

The findings were made public by Dr. Douglas I. Hammer of EPA's National Environmental Research Center in North Carolina. The report is based on a 1972 survey of respiratory illnesses among children living in four New York communities representing different levels of exposure to suspended sulfates, particulates, and sulfur dioxide.

Dr. Hammer said that this retrospective study showed that acute bronchitis was reported about 20 percent more frequently among children aged 1-12 exposed to these pollutants than comparable children not exposed. Excesses of croup and other lower respiratory infections were also found. He said differences in family size and composition, crowding, parental cigarette smoking or indoor air pollution could not explain the excess rates in the high exposure communities.

Indoor air pollution from gas stoves or space heaters has been mentioned as a possible risk fact for acute respiratory diseases. [illegible] significant differences or consistent trends were seen in the age group considered in this study, Dr. Hammer said.

"Repeated lower respiratory illnesses [illegible] children may well contribute to the development of chronic respiratory conditions in adult life," Dr. Hammer said in commenting on the study. "Therefore, it is important that we do everything possible to reduce this early risk."

Dr. Hammer's colleagues in this project were F. J. Miller, [illegible] A. G. Stead, M.S.; and Carl [illegible], Ph.D., and their research was part of EPA's CHESS program (Community Health and Environmental Surveillance System). CHESS is designed to evaluate existing environmental health standards, aid in the establishment of new ones, as well as document changes in health related to variations in air quality.

AIR POLLUTION AND CHILDHOOD LOWER RESPIRATORY DISEASE: EXPOSURE TO SULFUR OXIDES AND PARTICULATE MATTER IN NEW YORK, 1972

D.I. Hammer, M.D., F.J. Miller, M.S., A.G. Stead, M.S., and C.G. Hayes, Ph.D.

Introduction

Illnesses of the respiratory tract are the most common cause of morbidity in the United States and [illegible] or 50 to 80% of all childhood illnesses (1-4). Repeated lower respiratory tract illnesses in childhood may well contribute to the development of chronic respiratory disease in adult life (5). [illegible] in / England, Japan, and Russia have implicated ambient sulfur dioxide and particulate matter as a cause of increased respiratory morbidity in children (6-10). Prospective studies in New York and Chicago found increased acute respiratory disease morbidity associated with exposure to sulfur oxide and particulate air pollution among parents and their children (11,12). Recent retrospective surveys of acute lower respiratory disease in children living in U.S. western smelter communities found excess illness associated with sulfur oxide air pollution (13,14). Thus, there is epidemiologic evidence from several populations from different countries associating measured childhood respiratory morbidity with exposure to sulfur oxide and particulate air pollution.

Although New York City has historically experienced high levels of air pollutants resulting from fossil fuel combustion, ambient air quality has improved markedly during the past few years (15). However, in May 1972, when this survey was done, children living in the higher exposure communities had been exposed previously to high levels of sulfur oxide and particulate air pollution. We describe here a retrospective survey of acute lower respiratory illnesses among children in four New York communities representing an exposure gradient to sulfur oxide and particulate air pollution. The primary study hypothesis was that reported respiratory illness rates in the three high exposure communities would be higher than in the low exposure community.

Materials and Methods

Community Selection

Using historical air quality data (1944-1970) obtained from the New York City Department of Air Resources (NYC-DAR) and the Suffolk County (New York) Department of Health, in conjunction with census data and current information on house and property values, communities which were socioeconomically similar but different in air pollution exposure were selected. Communities were ranked on the

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*Presented by Douglas I. Hammer, M.D., at the American Medical Association Air Pollution Medical Research Conference, December 5-6, 1974, San Francisco, California. Dr. Hammer is an epidemiologist at the Environmental Protection Agency, Research Triangle Park, North Carolina.

basis of their exposure to sulfur dioxide and total suspended particulate matter. The rankings were relative in relation to the pertinent U.S. national primary air quality standards. Riverhead, Long Island, was designated the Low exposure community. The Howard Beach section of Queens and the Westchester section of Bronx were ranked as Intermediate exposure communities (16). Howard Beach was located close to the John F. Kennedy Airport, and its residents may well have been exposed to pollutants such as organic air pollutants and noise which were not monitored during this study and could not be accurately estimated during the previous years. Thus, Sheepshead Bay in Brooklyn, located geographically close to Howard Beach, but farther from the airport, was chosen as a third Intermediate exposure community. Detailed descriptions of the first three communities and the reasons for their selection have been published (15).

Assessing Air Pollution Exposure

CHESS air monitoring stations were established in Riverhead, Queens, and Bronx during 1970 and were located about 35 to 40 feet above the ground on tops of buildings. At each station, 24-hour integrated samples of sulfur dioxide (modified West-Gaeke method), total suspended particulates (high volume sampler), respirable particulates (cyclone separator, from 1972 on), suspended sulfates and suspended nitrates (chemical analysis of portion of total suspended particulate filter) were monitored on a daily basis. Dustfall was determined from monthly samples. A monitoring site was not established in Sheepshead Bay before this study was completed. Since we were primarily concerned with the effects of long-term exposure and since Sheepshead Bay is located near Howard Beach, current and historial aerometric data from the latter station were also used for Sheepshead Bay. Estimates of past exposures were derived from historical information supplied by NYC-DAR and the Suffolk County (New York) Department of Health. A complete description of the location of monitoring stations, the methodology for current monitoring, and the estimation of past exposures has been published previously (15).

Collection of Health and Demographic Data

In May 1972, elementary school children (grades 1-6) in each of the selected study communities were asked to take explanatory letters and questionnaires home to their parents. Socioeconomically similar school districts in each area were selected after consultation with local school officials and school principals. Children in three schools in each area were surveyed. The frequency of acute lower respiratory disease was ascertained by means of a School and Family Health Questionnaire which was completed by the mother or female guardian in each family when possible and returned to the school. Mothers were asked to answer the questions about lower respiratory disease for all children twelve years of age or younger who lived in the household.

The questionnaire inquired about the frequency of treatment by a physician for pneumonia, croup, bronchitis, or any chest infection other than these three during the three years prior to the time of questionnaire completion. Hence, all morbidity rates presented encompass the three year period from June 1969 through May 1972. Other information ascertained included hospitalizations for lower respiratory illnesses, name of children's current physician, history of

asthma diagnosed by a doctor, length of residence in the community, education of head of household as an index of socioeconomic status, parent's smoking status, race, family census, number of rooms per household, and use of a gas stove for cooking or a gas space heater in the home.

Data Analysis and Hypothesis Testing

As planned in the protocol, children with a history of asthma and all children with less than three years residence in the community were excluded from the analysis. Children with asthma are known to have a higher risk of respiratory illnesses than nonasthmatic children, as they did also in this study, and these results will be reported separately. Children with less than three years residence were excluded because they had been exposed to the community air for only a short time and no knowledge of their previous residence and hence, of their previous pollutant exposure, was available. Moreover, illness itself has been associated with migration and recently migrated families would be least likely to have established patterns of medical care within a community which could confound the effect of pollution with the effect of migration on reported doctor-diagnosed illnesses within a community. Black and white children were analyzed separately because a previous study had shown differences in reported acute lower respiratory disease morbidity patterns by race (17). Children under one year of age had to be excluded due to an error in coding instructions which made children under age one indistinguishable from children with missing information on age.

The major test hypothesis was that the frequency of reported physician treatment for "any lower respiratory illness," pneumonia, croup, bronchitis, and and "other" chest infections, appropriately adjusted, would be higher in the Intermediate exposure communities than in the Low exposure community. A combined disease category, "any lower respiratory disease," was constructed to include a child if he had either pneumonia, croup, bronchitis, or any deep chest infections other than the previous three ("other"). For each morbidity condition, two sets of analyses were done. In the first, the dependent variable was the percent of children reporting one or more episodes of each morbidity condition. In the second, the dependent variable was the percent of children reporting two or more episodes of each condition.

Each specific hypothesis was tested statistically in a general linear model for categorical data (18). This technique utilizes weighted regression on categorical data and allows estimation of each individual factor adjusted for all other factors in the model. Independent variables used in the model were age (three categories; 1 to 4, 5 to 8, 9 to 12), sex, and as an index of socioeconomic status, education of the head of household (2 categories: less than high school, high school or more). Therefore, there were forty-eight subpopulations in the model for each morbidity condition, viz. community exposure (4), age (3), sex (2), and education of the head of household (2). With approximately 48 subpopulations, the variance matrix used in the linear categorical model becomes unstable under two conditions: first, if more than a few cells have zero rates, and second, if less than 30-40 observations occur in any subpopulation. For the few times these constraints were violated, a simpler model was fitted whenever possible. Descriptive rates were direct-adjusted according to standard methods using all white nonasthmatic children with three or more years residence duration as the reference population (19).

During preliminary statistical analyses, uncommonly high rates of pneumonia were observed in Riverhead. These rates were found not to be due to a systematic error. Subsequent study indicated that about 40% of the children in Riverhead were attended by one of two doctors in the same office for their lower respiratory illnesses, whereas all other children in Riverhead were seen by over fifty different physicians. Furthermore, lower respiratory disease rates in Riverhead varied significantly when similar children from the first group were compared to those in the second group. In Queens, three physicians saw enough children (13 to 15%) to be compared to all other physicians and no differences in lower respiratory disease rates were found. Such comparisons were unnecessary in Bronx and Sheepshead Bay because no one physician saw enough children to permit one. Consequently, an additional independent variable was added to the model for white children, viz. "doctor in Riverhead," being the contrast of each morbidity rate between children seen by the first two doctors and those children seen by all other doctors in Riverhead. Rates of pneumonia and one or more episodes "any lower respiratory disease" and hospitalization were significantly higher among the children seen by the two previously mentioned doctors whereas rates of the remaining three morbidity conditions were similar. Because of smaller sample size, black children in Riverhead seen by the two doctors were excluded from all contrasts.

Results

Environmental Exposure

Estimated exposure to ambient pollutants for all study communities from 1960 to 1972 are summarized in Table 1. For the reasons previously mentioned, exposure in Sheepshead Bay was assumed to be similar to that of Queens. In Queens and Bronx, estimated annual sulfur dioxide levels were similar, ranging from about 175 to 425 ug/m^3 from 1960 through 1970 and about 40 to 50 ug/m^3 during 1971 and 1972. Average annual sulfur dioxide levels in Riverhead were about 25 ug/m^3 in 1971 and 1972 and were assumed to be quite low during 1960-1970, although measured data are not available. Estimated average annual total suspended particulate levels in Queens and Bronx ranged from about 85 to 195 ug/m^3 during 1960 to 1970 and from about 60 to 90 ug/m^3 in 1971 and 1972. Comparable total suspended particulate levels in Riverhead during 1971 and 1972 were about 35 ug/m^3 and no data were available for the period 1960 to 1970. Respirable suspended particulates measured only in 1972 were only slightly higher in Queens and Bronx than in Riverhead.

Suspended sulfate levels in Queens and Bronx ranged from about 9 to 20 ug/m^3 during 1960 to 1970 and were about 14 ug/m^3 during 1971 and 1972. Data from Riverhead indicated levels of about 10 ug/m^3 during 1971 and 1972. Annual average suspended nitrate levels in all three communities were low during 1971 and 1972, being lowest in Riverhead. When compared to children in Riverhead, children in Queens and Bronx were exposed to higher levels of sulfur dioxide and total suspended particulate matter until about a year and one-half before the survey. Suspended sulfate levels, which were also higher in Queens and Bronx during 1960 to 1970, remained moderately higher than those in Riverhead during 1971 and 1972.

Characteristics of Family Households

Response rates were good, being about 80% in all four communities. Characteristics of study families by community and race are given in Table 2. Ninety-five percent or more of all families, except whites in Riverhead (89%) lived in their community for three or more years. Although the median age of mothers or maternal guardians was similar in all communities, significantly fewer black families had both parents or guardians present. Black families in Riverhead had a lower proportion of heads of households with a high school or better education than any of the others. Parental cigarette smoking habits were comparable by race and community with females smoking somewhat less than males. Black families tended to have more persons per household and fewer rooms per household when compared to white families (Table 3). Riverhead had a lower proportion of households using either gas stoves or gas space heaters.

Lower Respiratory Illnesses in Children

As expected, the age distribution of children used in the analyses was comparable for both rates in all communities (Table 4). Morbidity rates generally decreased with age for both races, as expected. Among black children, bronchitis, croup, and "other" chest infections were reported in decreasing frequency and all conditions except pneumonia and hospitalization were reported more frequently in the more polluted communities than in Riverhead (Table 5). Among white children, bronchitis was reported a bit more frequently than croup and "other" chest infections (Table 6). Again, all conditions except pneumonia and hospitalization were reported more frequently in the more polluted communities, when compared to Riverhead. When compared to white children, black children had higher rates of pneumonia and lower rates of croup, bronchitis and "other" chest infections. Hospitalization rates were similar among blacks and whites. Patterns for two or more episodes were similar to those for one or more episodes for each condition.

Model adjusted morbidity rates for blacks and whites are shown in Table 7 and Table 8. Among black children, rates of "any lower respiratory disease," croup, two or more episodes of bronchitis and one or more episodes of "other" chest infections were significantly higher in the more polluted communities with relative increases ranging from about 1.7 for one or more episodes of "any lower respiratory disease" to about 3.2 for one or more episodes of "other" chest infections. Among white children, all morbidity conditions except pneumonia and hospitalization were significantly higher in the more polluted communities with relative increases ranging from about 1.1 for one or more episodes of croup to about 1.5 for one or more episodes of "other" chest infections. Bronchitis and one or more episodes of "any lower respiratory disease" were significantly only among children aged 5 to 12 years. When rates in Queens, Sheepshead Bay, and Bronx were contrasted, Bronx was lowest the few times that differences were significant (Table 8). Pneumonia and hospitalization rates were significantly higher in Riverhead, although the absolute rates for both conditions were low. [illegible] significant, morbidity rates were higher among males and among children from households with a high school or greater education.

Discussion

Reported lower respiratory disease morbidity was significantly higher among children exposed to ambient sulfur oxide and particulate air pollution than comparable children not so exposed. Rates of "any lower respiratory disease," croup bronchitis, and "other" chest infections were increased among black and white children residing in the more polluted communities (Tables 7, 8). Rates of pneumonia and hospitalization were significantly increased in the low pollution community only among white children and the absolute rates for both conditions in all communities were low. As expected, morbidity rates declined with age and were generally higher in males and among children from households with a high school or greater education.

Several other known determinants of childhood respiratory disease morbidity besides ambient air pollution were comparable among the communities for each race. Exposure to sidestream smoke has been associated with increased acute respiratory disease morbidity in children (20). The parental smoking habits were similar in all four communities and lower respiratory disease morbidity did not differ among children of smoking and nonsmoking parents. Family size and composition and household size did not differ among the communities. Age sex, and education of the head of the household were accounted for in the analysis.

Indoor air pollution such as oxides of nitrogen and carbon monoxide emanating from gas stoves or gas space heaters has been mentioned as a possible risk factor for acute respiratory disease. Although oxides of nitrogen have been linked to excess acute lower respiratory disease morbidity, evidence for such an association with carbon monoxide is currently lacking (22-24). Except for whites in Riverhead, from 90 to 98% of all households used a gas stove for cooking and/or a gas space heater (Table 3). Due to small sample size, only white, non-asthmatic, residentially stable children from households with a high school or greater education could be analyzed in a model testing the relationship of all respiratory morbidity categories as a function of the presence of gas stoves or space heaters in the household, age and sex. No significant differences nor consistent trends were found with regard to respiratory morbidity and the presence of gas stoves and/or spece heaters in the household. Thus, our data do not associate excess acute lower respiratory disease in children with such exposures. Perhaps if studies were restricted to persons who would be most likely to be exposed, viz. mothers or the person who cooks, an association might be found. Nevertheless, the difference in the presence of gas stoves or space heaters between Riverhead and the other three communities surely was not the cause of the excess morbidity observed in this study.

This study corroborates similar earlier observations of differences in morbidity reporting between blacks and whites and the possible explanations for such differences were discussed in detail in the previous report (17). In this study, when compared to white families, black families were larger, had only slightly less average living space within their respective community, were less educated only in Riverhead, but did have considerably fewer households with both

parents or guardians present, viz. about 56% for blacks versus 85 to 93% for whites. Patterns of reporting for blacks and whites were similar with regard to age and history of childhood asthma suggesting the difference was not a statistical artifact. Differences in medical care utilization could partially explain our results. In a recent U.S. national survey, nonwhites reported fewer physician visits than whites did for each family size category (25). Black white differences in pulmonary function and susceptibility to cigarette smoke have also been reported (26-28). Differences in recall, access to care, parental perception of illness, as well as other cultural or genetic differences all may be associated with the observed black-white disparity in reported morbidity. Although answers to these questions are of clinical and public health interest, this study was not designed to address them.

In this study, excess acute lower respiratory disease morbidity in children was associated with estimated exposure to average annual sulfur dioxide levels of about 38 to 425 ug/m^3, total suspended particulate levels of about 60 to 185 ug/m^3 and suspended sulfate levels of about 9 to 20 ug/m^3. Respirable particulates were 34 and 43 ug/m^3 in Queens and Bronx compared to 28 ug/m^3 in Riverhead, but data were available only for the five months of 1972 that the study encompassed. Since the study encompassed up to twelve years of exposure for the oldest children and respirable particulates are not related to total suspended particulate matter in a simple linear fashion, it is not possible to estimate a threshold effects level for respirable particulates per se. Recent work has linked excess morbidity with exposure to annual total suspended particulate concentrations of about 100-140 Ug/m^3 and suspended sulfate levels of about 13-14 ug/m^3 compared to respective levels of about 75 ug/m^3 and 10ug/m^3 in the lower exposure community, and average annual sulfur dioxide levels were only 25 ug/m^3 in both communities (17). However, information regarding lower respiratory disease and exposure to average annual particulate levels of about 60 to 89 ug/m^3 compared to about 35 ug/m^3 is not currently available. Therefore, a "best judgment" estimate of the levels required to produce effects would be exposure to average annual levels of about 175 to 250 ug/m^3 of sulfur dioxide, 85 to 110 ug/m^3 of total suspended particulates and about 13-14 ug/m^3 of suspended sulfates. Such concentrations are certainly within the range of those estimated from earlier studies (6, 7, 13, 14). Since sulfur dioxide levels have decreased considerably in Queens and Bronx while total suspended particulate levels remain near the Federal standard, a replication of this survey would provide an opportunity to further distinguish between the effects of these two pollutants.

Summary

Acute lower respiratory disease in children aged 1 to 12 years was surveyed retrospectively by questionnaire among parents in four New York metropolitan communities representing different exposures to sulfur dioxide, particulate matter and suspended sulfates. Morbidity patterns were similar with regard to age for blacks and whites, but pneumonia was more frequent and bronchitis and "other" chest infections less frequent among blacks when compared to whites in each community. Rates of "any lower respiratory disease" (a combined category), croup, bronchitis, and "other" chest infections were significantly higher among black and white children residing in the higher pollution exposure communities. Pneumonia and hospitalization were significantly higher only among white children in the low exposure

community but the absolute rates were low for both conditions in all communities. Differences in family size and composition, crowding, parental cigarette smoking or indoor air pollution due to gar stoves or gas space heaters could not explain the morbidity excesses in the high exposure communities. The "best judgment" estimate of the average annual pollutant concentrations associated with excess childhood respiratory morbidity in this study was 175 to 250 ug/m^3 of sulfur dioxide, about 85 to 110 ug/m^3 of total suspended particulates, and about 13-14 ug/m^3 of suspended sulfates.

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Table 1

Estimated Average Annual Pollutant Exposure Levels for Three* Study Communities in the New York Metropolitan Area, 1960-72

Pollutant	Community	Estimated Average Annual Concentration, ug/m^3**				National Primary Air Quality Standard, Annual Average
		1960-67	1968-70	1971+	1972+	
Sulfur Dioxide	Riverhead	Not Available		23	22	80 ug/m^3 (0.03 ppm)
	Queens	~425	~175	51	50	
	Bronx	~400	~250	51	38	
Total Suspended Particulates	Riverhead	Not Available		34	36(32)[a]	75 ug/m^3 (Geometric Mean)
	Queens	~185	~85	63	89(79)	
	Bronx	~175	~170	86	60(55)	
Respirable Suspended Particulates	Riverhead	Not Available			28	None
	Queens				34	
	Bronx				43	
Suspended Sulfates	Riverhead	Not Available		10	11	None
	Queens	~20	~9	13	14	
	Bronx	~18	~15	14	14	
Suspended Nitrates	Riverhead	Not Available		2	2	None
	Queens			4	3	
	Bronx			4	3	

*Although aerometric data were not available, it was assumed that exposure in Sheepshead Bay, the fourth study community, would be most similar to Queens community of Howard Beach since it is geographically continguous to it.

**Adapted from Table 5.1.4, reference 15.

+Measured data

[a]Geometric mean in parentheses

Table 2

Characteristics of Study Families, by Community and Race

Community (Number of Households)	Race	Three or More Years Residence Duration, %	Median Age of Mothers, years	Both Parents or Guardians Present, %	Fathers Completing High School, %		Current Cigarette Smokers, %+	
					≤ 39 yrs	≥ 40 yrs	Mothers	Fathers
Riverhead (275)	Black	95	33	59	35	27	41	49
Q,B,S* Combined (302)		96	34	53	79	67	45	49
Riverhead (620)	White	89	34	85	78	46	45	51
Queens (991)		95	35	93	82	73	49	48
Bronx (957)		96	35	87	72	58	46	53
Sheepshead Bay (1066)		97	35	88	85	83	50	50

*Queens, Bronx, and Sheepshead Bay combined due to small sample size

+Direct adjusted for age and education

Table 3

Characteristics of Households, by Community and Race

Community	Race	Number of Persons per Household**		Number of Rooms per Household		Presence of Gas Stove or Gas Space Heater	
		Median	Four or More	Median	Six or More	Stove Only	Heater Only or Both+
Riverhead	Black	4	51%	5	45%	77%	12%
Q,B,S* Combined		3	42%	5	20%	93%	4%
Riverhead	White	3	32%	6	64%	54%	5%
Queens		3	22%	7	87%	71%	25%
Bronx		3	23%	5	42%	87%	11%
Sheepshead Bay		2	13%	5	44%	90%	8%

*Queens, Bronx, and Sheepshead Bay combined due to small sample size

**Excluding parents or guardians

+Less than 1% of households had a gas space heater only

Table 4

Age Distribution of Nonasthmatic Children with Three or More Years of Residence, by Community and Race+

Community	Race	Number of Children			Total
		1-4 years	5-8 years	9-12 years	
Riverhead	Black	102	218	224	544
Q,B,S* Combined		85	218	287	590
Riverhead	White	220	495	481	1196
Queens		328	792	720	1840
Bronx		273	769	718	1760
Sheepshead Bay		310	793	726	1829

*Queens, Bronx, and Sheepshead Bay combined due to small sample size

+Excludes children with missing information on morbidity, age, race, sex, education of head of household, or duration of residence (3% of sample for whites and 11% of sample for blacks)

Table 5

Three Year Frequency of One or More Episodes of Morbidity
Among Black Children in New York by Age and Community

Community+	Morbidity Condition	Age-specific Rates, %*		
		1-4 years	5-8 years	9-12 years
Riverhead	"Any Lower Respiratory Disease"	20.3	16.4	6.9
Q,B,S Combined+		32.9	18.8	18.1
Riverhead	Pneumonia	10.1	10.3	2.9
Q,B,S Combined		16.5	5.8	4.5
Riverhead	Croup	3.8	2.4	1.7
Q,B,S Combined		11.8	5.5	6.6
Riverhead	Bronchitis	8.9	6.7	2.9
Q,B,S Combined		12.9	8.2	6.6
Riverhead	"Other Chest" Infections	2.5	1.2	0.6
Q,B,S Combined		4.7	3.7	3.8
Riverhead	Hospitalization	7.6	6.1	1.1
Q,B,S Combined		1.2	0.9	1.3

*Rates of "any lower respiratory disease," bronchitis, and pneumonia declined significantly with sex or education of head of the household.

+Queens, Bronx, and Sheepshead Bay combined due to small sample size.

Table 6

Three Year Frequency of One or More Episodes of Morbidity Among White Children in New York by Age and Community

Community	Morbidity Condition	Sex and Education of Head of Household Adjusted Rates, %*		
		1-4 years	5-8 years	9-12 years
Riverhead	"Any Lower Respiratory Disease"	30.8	27.6	11.2
Queens		38.6	36.7	28.4
Bronx		34.2	31.0	22.9
Sheepshead Bay		35.6	39.9	24.5
Riverhead	Pneumonia	9.5	3.8	1.5
Queens		2.1	2.0	2.2
Bronx		2.8	4.9	2.8
Sheepshead Bay		1.7	2.4	2.6
Riverhead	Croup	10.6	11.4	1.9
Queens		16.6	13.9	6.4
Bronx		12.9	9.9	6.2
Sheepshead Bay		15.1	13.7	5.7
Riverhead	Bronchitis	15.8	15.4	7.2
Queens		14.7	15.4	13.1
Bronx		17.2	17.6	10.7
Sheepshead Bay		12.5	18.1	11.8
Riverhead	"Other" Chest Infections	5.0	4.5	4.4
Queens		15.0	14.4	12.4
Bronx		11.0	8.4	9.2
Sheepshead Bay		16.7	15.9	11.4
Riverhead	Hospitalization	7.3	2.8	0.8
Queens		1.8	1.0	0.4
Bronx		2.3	1.5	0.6
Sheepshead Bay		1.6	1.5	0.6

*All conditions decreased significantly with age, all but pneumonia and hospitalization were reported more frequently among households with a high school or greater education; "any lower respiratory disease," croup, and pneumonia were reported more frequently among males.

Table 7

Three Year Frequency of Morbidity: Model Predicted Rates for Black Children Aged 1 to 12 Years, by Morbidity Condition and Number of Episodes

Morbidity Condition	Number of Episodes	Model Predicted Rates, %*		High/Low Polluted Community ($p < 0.05$)
		Riverhead	Queens, Bronx, and Sheepshead Bay Combined**	
"Any Lower Respiratory Disease"	≥1	14.0	23.2	1.66
	≥2	4.9	9.4	1.92
Pneumonia	≥1	No significant difference between communities		
	≥2			
Croup	≥1	2.8	7.3	2.61
	≥2	0.1	2.8	28.0
Bronchitis	≥1	No significant difference between communities		
	≥2	1.0	3.0	3.0
"Other" Chest Infections	≥1	1.3	4.2	3.23
	≥2	No significant difference between communities		
Hospitalization	≥1	Rates too low to fit model		
	≥2			

*Restricted to nonasthmatic children with three or more years residence duration; adjusted for age, sex, and education of head of household for "any lower respiratory disease," pneumonia, and bronchitis; adjusted for age for croup and "other" chest infections.

**Queens, Bronx, and Sheepshead Bay combined due to small sample size..

Table 8

Three Year Frequency of Morbidity: Model Predicted Rates for White Children Aged 1 to 12 Years, by Morbidity Condition and Number of Episodes

Morbidity Condition	Number of Episodes	Model Predicted Rates, %*		High/Low Polluted Community ($p < 0.05$)
		Riverhead	Queens, Bronx, and Sheepshead Bay Combined**	
"Any Lower Respiratory Disease"	≥1[a]	27.2	32.0	1.18
	≥2[b]	13.4	16.3	1.22
Pneumonia	≥1	5.7	2.6	0.46
	≥2	1.7	0.7	0.41
Croup	≥1	8.4	9.1	1.08
	≥2	No significant difference between communities		
Bronchitis	≥1[a]	11.6	14.2	1.22
	≥2[a]	4.2	5.2	1.24
"Other" Chest Infections	≥1	7.7	11.6	1.51
	≥2	3.9	5.7	1.46
Hospitalization	≥1	2.8	1.5	0.54
	≥2	Rates too low to fit model		

*Restricted to nonasthmatic children with three or more years residence duration; adjusted for age, sex, education of head of household, and doctor in Riverhead.

**No significant difference among three communities for pneumonia, croup, bronchitis, and hospitalization; Queens and Sheepshead Bay equal to each other and higher than Bronx for "other" chest infections; Sheepshead Bay > Queens > Bronx for "any lower respiratory disease" among 5-8 year olds only.

[a]significant among children aged 9-12 years only

[b]significant among children aged 5-12 years only

Appendix D-1.2

Exhibit shown during interview

THE WHARTON SCHOOL
of the
UNIVERSITY of PENNSYLVANIA
PHILADELPHIA 19174

VANCE HALL CS
3733 Spruce Street

MANAGEMENT AND BEHAVIORAL
SCIENCE CENTER

January 21, 1975

Dorothy Hayes Kane, M.P.H., M.U.S.
14 Charlton Hill
Hamden, Conn. 06518

Dear Ms Kane:

In response to your request of 13 January for reprints of publications pertaining to relationships of air pollutants to child health, I have enclosed a list of publications some of which may be of interest to you. They pertain particularly to health consequences of atmospheric chemicals, and also to the smoking-health problem. Several reprints are enclosed.

Only some of our publications, thus far, relate to effects of pollutant chemicals on child health. We have reported some findings regarding congenital malformations mortality rate. We have results, not yet published in the indexed literature, that pertain to birth rate and to mortality rate for infants less than one year of age. Some of the material pertains to a theory that certain air pollutants, particularly SO_2 and NO_x, are mutagenically hazardous. Statistical findings are consistant with this theory.

I hope the enclosed material may be of some assistance to you.

Please note that my address differs from the address you used.

Sincerely,

Richard J. Hicley

Enclosures
RJH/ccb

EXHIBIT B

Ecological Statistical Studies Concerning Environmental Pollution and Chronic Disease

RICHARD J. HICKEY, DAVID E. BOYCE, EVELYN B. HARNER, AND RICHARD C. CLELLAND

Abstract—Ecological statistical studies employing methods of multivariate analysis, based on a radiomimetic or mutagenic hypothesis, have yielded a number of statistically significant multiple regression equations in which concentrations of environmental chemicals, largely air pollutants, predict annual mortality rates for major categories of cancer and heart disease, as well as for congenital malformations, for populations of 38 metropolitan areas of the United States. Median age of these populations was also predicted statistically. Squares of multiple correlation coefficients R in excess of 0.5 were frequently obtained for these equations along with related t and F statistics of suitable magnitude. A new computer program for optimal regression analysis was employed in the studies.

Among the chemical predictors whose atmospheric concentrations are frequently found positively correlated with mortality rates are SO_2, NO_2, and particulate sulfate. Among frequently recurring negatively correlated predictors are Cu, Cd, and Ti. Evidence regarding whether SO_2 and NO_2 may be considered as mutagenic hazards to life is discussed, as are some potentially relevant biochemical functions of the metals.

I. Introduction

THE relationship of atmospheric pollution to human health has been a matter of concern in many parts of the world for many years. However, obtaining or retrieving a healthful livable environment has only recently become an important political and rhetorical issue, even though pollution of air, rivers, lakes, and oceans has been occurring with varying degrees of severity for many years. It is, or appears to be, more "profitable" to pollute than not to pollute. The alternative to discarding waste into the air or water is to build waste recovery and disposal facilities, often at considerable expense, or to recycle materials if possible [1], [2]. The discussion of Ayres and Kneese [1] on internalizing these externalities will not be received with enthusiasm in many quarters. Benefit cost types of analyses have been considered by various authors, such as Netschert [3], but there are problems in estimating the health costs of pollution until it is clearly established what the health effects of environmental pollution really are.

Many industries, and also many governmental bodies, employ waste disposal to the environment causing pollution in the interests of "economy" [1]. It is widely considered "cheaper" to run sewage into a river, killing fish, ruining oyster beds, and spoiling recreational facilities than it is to build and operate effective sewage disposal plants. This economy is difficult to explain, for example, to fishermen and oyster men who may be put out of business or forced to move. To some, this "trade-off," with government approval or inaction, may seem a minor price to pay for conservation of governmental or industrial funds. This ill treatment of fishermen may be of relatively minor importance compared with the prospect that atmospheric pollution, along with the influences of other environmental chemicals, may be functionally involved in hastening or inducing the occurrence of certain major chronic diseases of man, e.g., cancer, heart disease, stroke, and others whose mortality rates are positively correlated with chronological age, and may also relate to congenital malformations and the process of senescence. In 1964, a Presidential Commission [4] estimated that the annual cost of cancer, heart disease, and stroke in the United States exceeded $50 billion. This is, of course, apart from considerations of personal hardship and suffering.

A major purpose of our studies is to attempt to determine the extent to which environmental chemicals, along with other broad classes of environmental variables, e.g., demographic, ethological, climatic, and economic, are associated statistically with chronic diseases. Of equal or greater importance is the objective of investigating experimental evidence and theory in biology and biochemistry, particularly in molecular biology and ecological genetics, in an attempt to identify the mechanisms underlying the more important statistical results. Statistical correlations alone, of course, do not and cannot establish causality.

There have been a number of famous, or rather infamous, incidents of atmospheric inversions or other stagnations in which unusually severe episodes occurred briefly, for several days, resulting in [illegible] the mirror, or ill. Several episodes recorded to date include the Donora, Pa., tragedy of 1948, the episode in the

Manuscript received June 10, 1970. This study was supported by a Grant from Resources for the Future, Inc., by a Special Project Grant from the Council for Tobacco Research-U.S.A., by Public Health Service Institutional Grant [illegible] and by Public Health Service Grant [illegible] to the Regional Science Research Institute, Philadelphia, Pa. This paper was presented at the 1970 IEEE International Geoscience Electronics Symposium, Washington, D. C., April 14–17.

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For removed journal article please refer to the following reference:
(includes pp. 220-237)

ECOLOGICAL STATISTICAL STUDIES CONCERNING ENVIRONMENTAL POLLUTION AND CHRONIC DISEASE

by
R. J. HICKEY, D. E. BOYCE, E. B. HARNER, AND R. C. CLELLAND

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Appendix D-1.3

Exhibit shown during interview

Smoke Hurts Infants

LONDON (AP) — The chances of infants contracting pneumonia or bronchitis in their first year of life are nearly doubled if both their parents smoke, according to research by a team of British doctors.

"A picture has emerged of a serious risk to infants in the first year of life from exposure to their parents' cigarette smoke," the doctors reported in the current issue of the Lancet, a medical journal.

The team from St. Thomas' Hospital in London and the London School of Hygiene and Tropical Medicine reported it studied 2,500 children over five years.

They found that pneumonia or bronchitis can still be fatal to young children even when the illness receives prompt attention.

For removed journal article please refer to the following reference:
(includes pp. 239-242)

INFLUENCE OF PASSIVE SMOKING AND PARENTAL PHLEGM ON PNEUMONIA AND BRONCHITIS IN EARLY CHILDHOOD

by

J. R. T. Colley, W. W. Holland, and R. T. Corkhill

Reprinted from THE LANCET
Saturday, 2 November 1974
pp. 1031-1034

Appendix D-2

Sample of letter (with enclosure) sent to active practicing pediatricians in 3 "Dirty Air" and 9 "Clean Air" cities and towns in Connecticut, as follows:

"Dirty Air" area	"Clean Air" area
New Haven	Avon
Orange	Manchester
West Haven	New Milford
	Rockville
	Sharon
	Simsbury
	South Windsor
	Torrington
	Winsted

Dorothy Noyes Kane, M.P.H., M.C.S.

14 Charlton Hill, Hamden, Connecticut 06514: Telephone (203) 248-9544

7 April 1975

John T. Doe, M.D.
1 Main St.
Dirty Air Town, Ct.

Dear Doctor Doe,

Can you possibly allocate twenty minutes sometime in the next week or so to talk to a doctoral candidate (PhD) who is working in a dissertation dealing with air pollution and children?

I assume that your interest in the subject of the environment runs deep: so does mine (I initiated and headed an Environmental Studies Center at Southern Connecticut State College a couple of years ago, since which time I've been casing the world's biomedical literature pretty extensively and talking with researchers in the field).

Now I need to know how clinicians assess the situation, which is why I'm turning to you for help. If you will give me the benefit of your views, I shall be most grateful.

Sincerely,

Dorothy Noyes Kane

P.S. We also share an interest in children, as the enclosed review of a book of mine--published under my former married name--testifies. Incidentally, I've never met the Yale reviewer.

future of science. What they think about the world of today, and what the world of tomorrow will be or can be like cannot be lightly passed aside either by individuals or by governments.

YOUR CHILD STEP BY STEP TOWARD MATURITY by Dorothy Noyes Sproul. Doubleday & Co., Inc., Garden City, New York. 1963. 274 pp. $4.50.

Reviewed by: IRA S. GOLDENBERG

What are the prerequisites for an author of a volume on child rearing? A medical degree and extensive experience as a pediatrician? Prolonged exposure to the problems of children as a social psychologist? Or a rational knowledge of one's own experiences "just" as a parent? Dorothy Noyes Sproul is neither a physician nor a sociologist, but rather a former advertising executive who brought two children through the rigors of childhood and has gained insight and wisdom which she now set down in this thoughtful and fact-filled book. Her syndicated column dealing with the health problems of youngsters has been widely circulated and the present book represents a distillation of much of the material from these columns.

Mrs. Sproul has analyzed the health problems of children and has done a remarkably able job of conveying in a straight-from-the-shoulder manner the importance of preventive medicine, including the ever present threat of accidents—childhood's greatest killer. A carefully administered initial dose of "safety vaccine" is recommended, with almost continual boosters as the child grows. The discussions related to a parent's role in developing intellectual, spiritual and emotional strength in the child are particularly valuable to all parents in this age of atomic unrest. This is a book for every parent, and is recommended unequivocally to all physicians who should, in turn, recommend it to all parents and parents-to-be.

The well established Dr. Spock has been joined by an individual who expresses herself with brevity and commonsense and recognizes above all that we should listen to children more than we usually do. Mrs. Sproul's introduction states that, "Children are our greatest asset, our greatest natural resource. Without them, no tomorrow has any significance." Her book forcefully tells how to preserve this asset for a greater tomorrow that sometimes seems impossible today.

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Appendix D-3

AMA Guide offered at conclusion of interview

Appendix E

Presentation before the 142nd national meeting of the American Association for the Advancement of Science

Boston, 22 February 1976

POLLUTION AND THE AMBIENT AIR OF SMALL CHILDREN

In the fifteen minutes allotted to me, I'm going to do several things. First, I shall try to outline the ways in which small children differ from adults in their reaction to air pollutants: this will embrace biologic, metabolic, and behavioral distinctions. This is the group I shall be talking about--the 20 million youngsters in the U.S. under 5 years of age, some at greater risk than others because of area of residence, socioeconomic status, genetic inheritance, or race.

(Slide: Pedriatric Population at High Risk)

Next, I shall try to highlight what we think we know about the action of pollutants in the concentrations which exist most of the time in many parts of the U.S. today--but not during acute episodes--on the two systems of the body which would appear (at least from research to date) to bear most of the brunt from airborne noxae. These are the respiratory and central nervous systems. (There are, of course, other very vital systems affected, but these are out of bounds time-wise.)

Then I shall touch upon the reasons why I feel child-care people should be concerned about indoor as well as outdoor air quality, particularly in the light of certain human frailties, and counterailing societal forces. And, lastly, I shall try to persuade you that a "Mind-the-Iceberg" (MTI)--in contrast to a "Count-the-Bodies" (CTB)--approach is a much sounder way of preventing disease, and a much more humane and economical way of producing physically and mentally healthier bodies destined for a stronger, tougher adulthood.

My text, as preachers are wont to say, comes from a recent lead article in Lancet[1] on the subject of subclinical lead poisoning: "Thinking in black and white terms is out of date," the author says; it's an especially important grey area.

No need to tell this audience that children aren't just small adults. In a certain sense, though, this is a message that has only recently been appreciated in scientific circles. Standards for air quality have been set for grownups, not youngsters. Is such a policy wise, I wonder?

By and large, research which has focussed on the effects of air pollution in children has done so not because of interest in smallfry per se, but because youngsters are free of some of the variables which make the research purer. In short, children have been considered good epidemiologic subjects essentially because most of them don't smoke, and usually spend their lives in limited geographical areas where air monitoring can be more easily handled.

Children Differ from Adults

However, many studies and several years later, it now appears that there are perhaps seven ways in which children differ in their reactions to air pollutants, and this difference is distinctly not in their favor. Among these differences are the following:

(Slide)

1. Higher inspiratory flow rate at rest
2. Higher level of activity
3. Closer proximity of breathing zone to the ground
4. nature of play and activity
5. more mouth breathing
6. More frequent respiratory infections
7. Age-specific developmental factors

Children Inspire More Air

Let's consider the first two points: Per Unit of body weight, the young breathe in more air (and thus more pollutants) than adulsts. Add to this metabolic difference at rest the activity of small children, and you raise the inspiratory flow in an exponential manner; indeed, it is thought that, with exercise[2], pulmonary ventilation is increased by a factor of about 10.

Children Absorb More Pollutants

Now let's concentrate for a moment on the next two points in terms of the small child's world--let's consider the air 1 meter or thereabouts off the ground,

and let's think of the 20 million boys and girls as they play in this world.

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Not too much is known about the composition of air at or near ground level because devices used to measure air concentrations are rarely, if ever, placed at the height at which young children breathe. However, we do know that gravity forces many of the heavier and often more toxic particles to the ground (or floor). Cadmium is one example; others are asbestos, silica, plastics, etc. Some of these are respirable, some ingestible, some both.

In the case of lead (a highly potent neurotoxin), the concentration in ambient air varies inversely with altitude; in one sutdy, the concentration at 1.5 meters was twice that at 20 meters above the ground[3]. Other studies of street dirt and dust have revealed concentrations of lead far greater than those considered safe in paint by the Food and Drug Administration[4].

Similarly, the concentration of ozone is greater at a lower level. In fact, one study showed the concentration of ozone to be 4 times greater at 1 than at 2 meters[5]. Ozone, as you are probably aware, is a measure for a mixture of pollutants called photochemical oxidants, many of the individual components of which have not yet been identified. It acts synergetically with sulfur dioxide, and its standard is frequently exceeded in many parts of the country, including the northeast.

In addition to the fact that children's breathing zone is closer to the ground, there's the nature of their play: their dust-raising proclivity as they travel hither and yon, and their hand-to-mouth activity. Thus, besides the inhalation route, unmeasured amounts of polluted airborne dust and dirt are absorbed by children via the gastrointestinal tract. Unfortunately, besides having to cope with inspiring more pollutants than adults, children--it would seem-- have a greater rate of gastrointestinal absorption of such a toxin as lead (and possibly other undesirable chemicals as well), thus adding to their total body burden[6].

Children Have Filtering Problems

The next two points go hand in hand. Both exercise and blockage of the nasal passages increase mouth breathing, which decreases the efficiency of the normal filtering process. The mouth is a far less capable filter than the nose, and permits a much higher concentration of inhaled gaseous or particulate matter to reach the upper airways. Thus mouth breathing, coupled with a greater frequency of respiratory tract infections among the pediatric population, adds to the problems that small children must face in adjusting to, or coping effectively with air noxae.

Special Vulnerability of the Developing Organism

Which brings up the final difference: the immaturity of the young organism, whose systems and subsystems at various stages of development may be stressed or impaired in different ways depending upon the nature and degree of pollutant exposure, and upon each child's unique immunologic capability and sequential growth pattern. Unfortunately, there's a distinct possibility that some of the pollutant insults may prove irreversible.

Children's Lungs

What does air pollution do to children's respiratory tracts? Essentially it irritates the membranes lining the respiratory tract, and causes some degree of bronchoconstriction. In turn, the normal self-cleaning mechanism is affected and lung function depressed. Though the degree to which this occurs and persists under different exposures and for different periods of time is unresolved, impaired ventilatory performance at an early age is considered by many to be a risk factor for later respiratory troubles, including chronic bronchitis and emphysema. Dirty air appears to increase the incidence of acute lower respiratory tract diseases--such as bronchitis-- in children. It may contribute to more pediatric upper respiratory tract disease (and perhaps in children's families as well). Also, dirty air aggravates asthmatic and cardiopulmonary conditions, and may increase susceptibility to bacterial and viral infections. Among the principal airborne agents involved in respiratory

tract troubles: sulfur dioxide, acid sulfates, particulates, nitrogen dioxide, ozone, and asbestos.

Children's Brains

Children's nervous systems may be damaged by certain chemicals, metals, and mixtures thereof. The prevelance of low level lead toxicity resulting in "undue" absorption of this metal--which seems to have a special affinity for children's soft tissues, for their blood, and their brains--is widespread, far more so than was recognized a few years back. Unfortunately, it is asymptomatic. In nationwide screening programs in both small-and larger-sized communities, rural as well as urban young have been found at risk; so, too, have middle- and upper-income class youngsters, not just ghetto boys and girls living in dilapidated lead-paint-laden houses. From a number of recent studies comes evidence that low level lead is apparently associated with impaired brain function, resulting in neurological and psychological defects--such as a decrease in general cognitive, verbal, and perceptual abilities--and with behavior disorders, including hyperactivity[7-13]. Another dangerous metal--cadmium--may, with lead, exert a synergetic effect upon small children's central nervous systems.

Other Serious Problems

Time will not permit any but this fleeting allusion to the potentially carcinogenic, mutagenic, and teratogenic properties of a number of airborne agents, about so many of which--including their conversion products--we know too little as of this moment. It seems appropriate here to be reminded that cancer is the leading disease cause of death among children (accidents being the biggest killer).

Indoor Pollutants

Children don't necessarily get away from noxious chemicals when they go indoors, where--as a matter of fact--they may spend more than three-quarters of their day. Indeed, though the concentration of pollutants may be about the same indoors as out (depending upon

such factors as building structure, season of the year, etc.), air quality may actually be worse at home or school or day care center. (Floors and window-sills are special danger spots.)

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What are the contaminants lowering air quality indoors? The same as those outdoors, with perhaps more carbon monoxide, nitrogen dioxide, and various hydrocarbons--from tobacco smoke, from poorly vented heating and cooking equipment, and from aerosol sprays. Little children (including embryos and fetuses) should shun parents, particularly mothers, who smoke. (The dangers are so serious, so numerous, and so well documented.) Children should also interdict the use of aerosol sprays for whatever purpose, and wherever: in bathroom, kitchen, play room, hobby room, etc.

If an embryo opts to become a full-term, normal weight, well-formed newborn, it's wise for him or her to choose (among other attributes) a mother who eschews cigarettes. He or she needs non-smoking parents, too, during infancy and the very early years, if the goal is to stay clear of more than the customary share of serious respiratory ills (as has been suggested by several recent reports[14, 15]). At the same time, the hep embryo may be well advised to have mother checked out blood-lead-wise, for lead--like certain hydrocarbons --is know to cross the placental barrier. Incidentally, with reference to tobacco, recent studies also indicate that sidestream smoke from the burning of cigarettes contains compounds in higher quantities than in the mainstream smoke inhaled by the smoker[16].

Concern for Environment-Related Morbidity

This brings me to what I think child-care people should be into: a greater concern for the effects of dirty air on children's physical and mental health and wellbeing---a concern which, if translated first into an awareness of the problem, and then into conjunction with the decision-making process--could prove a real boon in terms of upgrading air quality. Let's face it, doctors are indispensable after the fact: their orientation is by and large

therapeusis for disease. While pediatricians are by training perhaps more prevention-minded than some of their colleagues, their education to date has not overexposed them to a recognition of environment-related morbidity. And, since there's no vaccine available for the troubles caused by dirty air, what recourse do they have? In their defense, they're heavily pressured by the demands of their patients' present illnesses: with a few exceptions they don't think of their small patients in terms of what they may become a decade or more from now when the internists will take over. Furthermore, community involvement isn't their long suit. Well, if doctors in general don't see clean air as a concern of theirs, what about the public health profession? Isn't clean air their responsibility?

I'm a member of this group, but a card-carrying maverick. As a result, I'm not addicted to what I refer to (rather ingloriously) as the party line, or a kind of rigid tunnel-type mind set. To put it in simple jargon, epidemiologists (as a result of their training) are a bit hung up on the criterion that--if you can't actually count the bodies--you can't prove what caused the damage. Furthermore, conventional wisdom isn't yet too comfortable with the multifactorial concept of disease; so, if you can't isolate the individual etiologic factor, you have no recourse but to continue to concentrate on sympton palliation.

Minding the Iceberg: The Preventive View

There's a small group of us, though, who subscribe to the "Mind-the-Iceberg" as opposed to this "Count -the-Bodies" view. We say, when you see its tip, you're pretty sure there's a great deal more beneath the surface, and what's below may be--I repeat just may be--even more dangerous.

(Slide)

You can't see it all, but it won't go away simply by ignoring it. If you keep probing, perhaps you can fathom its depth and density, and guessimate its potential for subsequent harm. (In the case of air pollution, the probing might very advantageously start at the ground or floor level, and thus might embrace a more realistic picture of present air quality standards from the pediatric position. This could involve possible reassessment in terms of dose-response

relationships which up to the present have failed to include a consideration of such parameters as age, height, and activity.)

Differences between MTI and CTB People

MTI people evaluate risk quite differently from the CTBers. What may seem a fair risk to a CTBer is often considered a dangerous risk by an MTIer. This is particularly true in evaluating risks for children, since they are admittedly highly susceptible to insults from air pollutants for the seven reasons already referred to.

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MTIers also have a different concept of the importance of optimal health, and especially of the importance of achieving such a state in adulthood by providing optimal environmental conditions during the developing years of infancy and childhood (and before, including the embryonic period). In short, MTIers opt for the long- as opposed to the short-term view, in assessing pollution's potentially traumatic toll over time.

Then, MTI people see the goal of optimal health not as an impossible dream, but as a more humane, socially desirable, and sounder economic goal, for they are mindful that prevention time and again has been proved infinitely cheaper than treatment and rehabilitation.

This is why MTIers advocate wider safety margins for many pollutants, and the setting of standards for more pollutants now unregulated like certain small particles and conversion products. Further, they believe that a demonstration of freedom from toxicity should be an obligation of industry, rather than that proof of incontrovertible harm of the "Count-the-Bodies" or gross pathology variety should be demanded of health professionals before corrective action is taken.

It's possible, of course, that we "Icebergers" are hounded by unfounded fears and that--unlike "Bodies" folk--we magnify potential dangers beyond all reason, and tend to fantasize about bad guys in academic robes whose judgments can be swayed by oilmen's grants. Quite to the contrary, my belief is that MTIers are actually hard-nosed realists. We all know

there's a limit to the dollars, rats, and time available to find answers to this incredibly complex maze of atmospheric gases, vapors, and aerosols, and their actions, interactions, and additive and synergetic effects, but erring on the side of prudence seems to MTIers to be the far sounder procedure not only as a means of improving the quality of life but of the GNP as well. (Coincidentally, we appreciate that a desirable spinoff of Project Air Clean-up is thousands of new jobs.)

Thus, in sum, I believe that MTIers are grey area people. We tend to concur in the view proffered by a prominent member of the Royal Society of Medicine that we haven't sufficiently considered the implications (as regards prevention) of the bronchitic child being father to the bronchitic man. With this in mind, consider the savings in terms of future productivity--of the many man and woman hours and years of productive labor gained if chronicity with its attendant disability is prevented from gaining a foothold in childhood. Consider, too, the economic, social and psychologic benefits if one possible cause of mental retardation could be eliminated.

Looking ahead a few years (from 1976-1980) the Environmental Protection Agency has projected that--unless SO_2 standards are met--usually healthy children will suffer 1.5-2.5 million more acute respiratory tract disorders like croup, bronchitis, pneumonia; and and asthmatic children will experience 10-20 million more unnecessary attacks[17]. Based on National Bureau of Standards and Center for Disease Control surveys[18], if exposure to lead is not curtailed, 1/2-1 million more children--in addition to the 600 thousand already seriously affected--will by 1980 show dangerously elevated lead levels, with all that this implies in terms of central nervous system damage.

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What of the Future?

It's not all doom and gloom for the MTI position. I'd like to conclude this brief resume by mentioning several Great Awakenings on the political front at the Federal level which would seem to augur well. I refer to the 5-year Forward Plan for Health for the period 1977-1982

just announced by the Department of Health, Education, and Welfare, which emphasizes the expansion of environmental and occupational health as major thrusts in a preventive health strategy to reduce the incidence and costs of disease.

> The Nation cannot afford a gap in its environmental health capacity. The consequences of hazardous environmental conditions and changes will be paid , for the most part, not by us but by our children and future generations.

Indeed, Dr. Theodore Cooper (Secretary for Health at the Department) believes that any national preventive health effort visualized in the Forward Plan must begin with the effect on children.

Also, I refer to a position statement from the June 1975 National Conference on Preventive Medicine:

> When there is partial, but not necessarily total evidence that a given environmental factor or situation is a cause of morbidity and mortality, decisions with respect to the implementation and emphasis of preventive measures must be weighed in prudence on the side of human beings involved rather than waiting for total scientific evidence.

Lastly, EPA has just announced its plan to increase consumer participation in the decision-making process. I hope that many non-smoking child-care people will become active participants, for consumer advocacy sorely needs persuasive leaders espousing the cause of greater pollution protection for our pediatric population. I hope, too, that--in addition to young professionals among these child-care people--there will be a reasonable sprinkling of caring grandparents. For this age group, because of de-development (rather than development) of its immune system, has a great deal in common with the very young in terms of immunocompetence and the ability to cope with toxic environmental agents[19].

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ABBREVIATIONS

AAA	American Academy of Allergy
AAP	American Academy of Pediatrics
AAAS	Americal Association for the Advancement of Science
ALA	American Lung Association
AMA	American Medical Association
APHA	American Public Health Association
ATS	American Thoracic Society
BCM	Bureau of Community Management
CDC	Center for Disease Control
CEH	Committee on Environmental Hazards
CSPI	Center for Science in the Public Interest
DEP	Department of Environmental Protection
EPA	U.S. Environmental Protection Agency
HEW	U.S. Department of Health, Education, and Welfare
LWV	League of Women Voters
NAS	National Academy of Sciences
NHLI	National Heart and Lung Institute
NRC	National Research Council
NERC	National Environmental Research Center
NICHD	National Institue of Child Health and Development
NIEHS	National Institute of Environmental Health Sciences
NIMH	National Institute of Mental Health
NIOSH	National Institute of Occupational Safety and Health
PHS	Public Health Service
PTA	Parent Teachers' Association
SIPI	Scientists' Institute for Public Information
WHO	World Health Organization

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